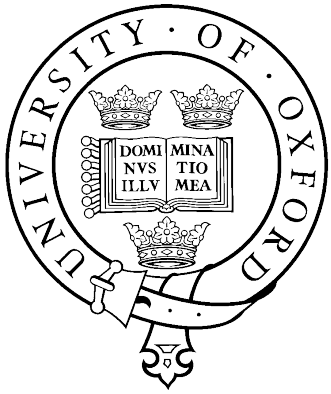
Income and asthma-related outcomes in children: a longitudinal study in the United Kingdom

Sophie Sanzan Diarra

St Cross College



A thesis submitted for the degree of

Doctor of Philosophy in Population Health

Hilary 2021

Acknowledgements

On pursuing a PhD, one embarks on an academic journey. They become all encapsulated by it, it becomes their companion. The PhD creates a web of intricate relationships, between not only the student and the thesis, but with many other persons. On that note I would like to thank all the people who helped me on my PhD journey: Firstly my brilliant supervisors Maria Quigley and Mara Violato, your patience knows no bounds and I am extremely grateful for your guidance and dedication to me all these years. To my family who kept asking "Is it done yet?", and silently smiling when I responded "Almost". To my friends, and colleagues in the Health Economics Research Centre and the wider department, who always encouraged me to just keep going and provided healthy distractions. I'd also like to thank Mona Bafadhel for her input on the topic of child respiratory health, and the graduate staff in the department for making administrative issues seamless. I am thankful for the studentship awarded to me by the Nuffield Department of Population Health and Medical Research Council, which allowed me to pursue this research. And a final thank you, to you who joined the journey a bit later, but gave me the final kick to the finish line.

Table of contents

Abstract……..……1

1. Introduction....3

1.1. Background....3

1.2. Income and asthma-related outcomes....5

1.3. Summary of income-related policies for families with children....7

1.4. Literature review....10

1.4.1. The association between income and child health....10

1.4.2. The association between socioeconomic status and asthma-related outcomes....19

1.5. How income determines child health outcomes....23

1.5.1. Transmission mechanisms....24

1.5.2. The direct effect of income....26

1.6. Changes in the child health-income gradient....26

1.7. Thesis objectives....27

2. Methods....30

2.1. The Millennium Cohort Study....30

2.2. Sampling in the MCS....31

2.3. Survey protocol....31

2.4. Survey content....32

2.5. Structure of the MCS datasets....33

2.6. Response rate....34

2.7. Study population....35

2.8. Asthma-related outcomes....37

2.9. Family income....39

2.9.1. Income for thesis data analysis....40

2.10. Other explanatory variables....42

2.10.1. A priori variables....43

2.10.2. Other potential explanatory variables....45

2.10.3. Mother and child health-related behaviour....47

2.10.4. Household and environmental factors....52

2.11. Statistical analysis....53

2.11.1. Theoretical model....53

2.11.2. Estimation methods....55

3. Cross-sectional analysis....57

3.1. Introduction....57

3.2. Methods....58

3.2.1. Analytical framework & model specification....58

3.2.2. Description of variables....59

3.2.3. Estimation strategy....60

3.2.4. Sensitivity analysis 62
3.3. Results 64
3.3.1. Overview of the data 64
3.3.2. Prevalence of asthma-related outcomes 72
3.3.3. The association between income and asthma-related outcomes 75
3.3.4. Changes in the gradient 82
3.3.5. The association between other factors and asthma-related outcomes 84
3.3.6. Sensitivity analysis results 89
3.4. Discussion 91
3.4.1. Summary of results 91
3.4.2. Age-specific prevalence of asthma-related outcomes 91
3.4.3. Income as a determinant of asthma-related outcomes in children 93
3.4.4. Other risk factors for asthma-related outcomes 95
3.4.5. Strengths and limitations 99
3.5. Conclusion 101
4. **Income and wheezing phenotypes 105**
4.1. Phenotypes in relation to asthma aetiology 105
4.2. Definitions of wheezing phenotypes 107
4.2.1. Pre-defined phenotypes 107
4.2.2. Categorisation by Latent Class Analysis (LCA) 111
4.2.3. The association between socioeconomic status and wheezing phenotype 117
4.3. Methods 119
4.3.1. Theoretical model 119
4.3.2. Model specification and estimation strategy 119
4.3.3. Sensitivity analysis 122
4.4. Results 123
4.4.1. Descriptive analysis 123
4.4.2. The association between income and wheezing phenotype 123
4.4.3. The association between other factors and wheezing phenotype 125
4.4.4. Sensitivity analysis 126
4.5. Discussion 131
4.5.1. Summary of results 131
4.5.2. Comparison with other studies 132
4.5.3. Other factors 133
4.5.4. Strengths and limitations 135
4.6. Conclusion 136
5. Panel data methods to analyse the association between family income and asthma-related outcomes 137
5.1. Introduction 137
5.2. The advantage of using panel data methods 138
5.2.1. Unobservable variables 138

5.2.2. Testing complicated behavioural hypotheses ... 138
5.2.3. More accurate predictions and inference ... 139
5.2.4. Determinacy over the sequencing of the causal mechanism ... 139
5.3. Methodology ... 140
5.3.1. Fixed effects methods ... 140
5.3.2. Random effects ... 144
5.3.3. Choosing between fixed effects and random effects ... 145
5.3.4. Theoretical model ... 146
5.3.5. Data ... 146
5.3.6. Modelling approach ... 147
5.4. Results ... 149
5.4.1. Approach 1: Using panel data methods ... 150
5.4.2. Approach 2 - Interaction between family income and MCS survey ... 162
5.5. Discussion ... 165
5.5.1. Summary of results ... 165
5.5.2. Strengths and limitations ... 168
5.6. Conclusion ... 169
6. Income-related inequality in child asthma-related outcomes ... 170
6.1. Introduction ... 170
6.2. Measuring income-related inequality in health ... 172
6.3. Concentration curves and the Concentration Index ... 176
6.3.1. Interpreting the Concentration Index ... 178
6.3.2. Shortcomings of the Concentration Index ... 178
6.3.3. Solutions to the shortcomings of the Concentration Index ... 180
6.4. Decomposition of the Erreygers' Index ... 183
6.5. Data ... 185
6.6. Analysis ... 186
6.7. Results ... 186
6.7.1. Erreygers' Indices ... 186
6.7.2. Decomposition analysis ... 188
6.8. Discussion ... 204
6.8.1. Summary of results ... 204
6.8.2. Strengths and limitations ... 206
6.9. Conclusion ... 207
209
7. Discussion of thesis ... 210
7.1. Introduction ... 210
7.2. Review of findings ... 210
7.2.1. The association between income and asthma-related outcomes ... 210
7.2.2. Steepening of the child health-income gradient ... 211

7.2.3. Income and wheezing phenotypes 212
7.2.4. Longitudinal analysis using panel data methods 213
7.2.5. Income-related inequality in asthma-related outcomes 214
7.2.6. Summary of all results 215
7.3. Comparison with other studies 215
7.4. The influence of other variables 217
7.5. Strengths and limitations of the thesis 219
7.6. Policy implications 220
7.6.1. Income welfare 220
7.6.2. Mother's health 223
7.6.3. Breastfeeding in the UK 225
7.7. Further research 227
7.8. Conclusion 228

List of abbreviations

ALSPAC	British Avon Longitudinal Study of Parents and Children
AME	Average Marginal Effect
AWE	Average Weekly Earnings
BAS	Benchmark Assessment System
BMI	Body Mass Index
CAPI	Computer Assisted Personal Interview
CI	Concentration Index
CPAG	Child Poverty Action Group
CPI	Chid Poverty Index
DWP	Department for Work and Pension
EI	Erreygers' Index
FE	Fixed Effects
GCI	Generalised Concentration Index
GCSE	General Certificate of Secondary Education
GMM	Generalised Method of Moments
HSE	Health Survey for England
ISAAC	International Study for Asthma and Allergies in Childhood
K6	Kessler Screening Test
LCA	Latent Class Analysis
ln	natural logarithm
LSAC	Longitudinal Study of Australian Children
LSDV	Least Squares Dummy Variable
MCS	Millennium Cohort Study
MEPS	Medical Expenditure Panel Survey
MLE	Maximum Likelihood Estimation
NHANES	National Health and Nutrition and Examination Survey
NHIS	National Health Interview Survey
NHS	National Health System
NLSCY	Canadian National Longitudinal Survey of Children and Youth
NVQ	National Vocational Classification
OECD	Organisation for Economic Cooperation and Development
OLS	Ordinary Least Squares
OR	Odds Ratio
PHE	Public Health England
PIAMA	Netherlands Prevention and Incidence of Asthma and Mite Allergy
PSID	Panel Study of Income Dynamics

RCOG	Royal College of Obstetrics and Gynaecology
RCPCH	Royal College of Paediatrics and Child Health
RE	Random Effects
RRR	Relative Risk Ratios
SES	Socioeconomic Status
SIDRIA	Italian Study of Respiratory Disorders in Childhood and the Environment
UK	United Kingdom
US	United States of America
WI	Wagstaff Index

List of figures

Figure 2.1: MCS research population 38

Figure 2.2: Card from MCS CAPI questionnaire showing weekly, monthly and annual income bands, after tax 41

Figure 3.1 Income quintile by age 65

Figure 3.2: Median and interquartile range for family income: Ever asthma 65

Figure 3.3: Median and interquartile range for family income: Wheezing in the last 12 months 66

Figure 3.4: Median and interquartile range for family income: Asthma medication use 67

Figure 3.5: Prevalence of ever asthma by age and income quintile 73

Figure 3.6: The prevalence of wheezing in the last 12 months by age and income quintile 74

Figure 3.7: Prevalence of asthma medication use by age and income quintile % (n) 75

Figure 4.1: Wheezing phenotypes classified by Martinez et al (1995) 107

Figure 4.2: MCS prevalence of wheezing phenotype 122

Figure 6.1: The Lorenz Curve for health 175

Figure 6.2: The Concentration Curve 178

Figure 6.3: Concentration curves: Ever asthma 207

Figure 6.4: Concentration curves: Wheezing last 12 months 208

Figure 6.5: Concentration curves: Asthma medication use 209

List of tables

Table 2.1: Survey elements MCS 1 and MCS 2 33

Table 2.2: A summary of study population and response in the MCS surveys 36

Table 2.3: MCS 2-5 asthma-related outcomes 38

Table 2.4: MCS 1 asthma-related outcomes 39

Table 3.1: MCS questions relating to asthma and wheezing 60

Table 3.2: Model specification for cross-sectional analysis 61

Table 3.3: Mean annual family income for all ages by income quintile 64

Table 3.4: Summary of explanatory variables from the MCS distributed by income quintile 70

Table 3.5: Prevalence of ever asthma by age and income quintile % (n) 73

Table 3.6: Prevalence of wheezing in the last 12 months by age and income quintile % (n) 73

Table 3.7: Prevalence of asthma medication use by age and income quintile % (n) 75

Table 3.8a: The association between income and ever asthma: Marginal effects 76

Table 3.8b: The association between income and ever asthma: Odds ratios 77

Table 3.9a; The association between income and wheezing in the last 12 months: Marginal effects 78

Table 3.9b: The association between income and wheezing in the last 12 months: Odds ratios 79

Table 3.10a: The association between income and asthma medication use: Marginal effects 80

Table 3.10b: The association between income and asthma medication use: Odds ratios 80

Table 3.11a: P-values for differences in the association of income across age: Ever asthma 83

Table 3.11b: Differences in the association of income across age: Wheezing in the last 12 months 83

Table 3.11c: Differences in the association of income across age: Asthma medication use 83

Table 3.12a: The association between lagged income and ever asthma: Marginal effects 90

Table 3.12b: The association between lagged income and wheezing in the last 12 months: Marginal effects 90

Table 3.12c: The association between lagged income and asthma medication use: Marginal effects 90

Table 3.13: The association between income and other factors: Marginal effects 102

Table 4.1: Wheezing phenotypes defined by age of onset using clinical definitions and using Latent Class Analysis 116

Table 4.2: Wheezing phenotypes defined in the base case analyses 121

Table 4.3: Model specification 122

Table 4.4: Wheezing phenotype by income quintile (N=9,921) 124

Table 4.5: The association between income and wheezing phenotype (base case): Marginal effects and RRRs 125

Table 4.6: Wheezing phenotype defined in sensitivity analysis 127

Table 4.7: The association between income and wheezing phenotype (cut-off age 7 for early remit): marginal effects and RRRs 128

Table 4.8: The association between income and wheezing phenotype (income at 9 months): Marginal effects and RRRs 130

Table 4.9: The association between income and wheezing phenotype (no family history of asthma sub-group): Marginal effects and RRRs 131

Table 4.10: The association between income and wheezing phenotype (with family history of asthma sub-group): Marginal effects and RRRs 132

Table 5.1: Asthma-related outcomes at each child age 148

Table 5.2: Longitudinal summary statistics of income and asthma-related outcomes 150

Table 5.3: Unadjusted FE and RE results (unweighted) 152

Table 5.4: Unadjusted FE and MLE results (weighted) 153

Table 5.5: Adjusted FE - Ever asthma 154

Table 5.6: Adjusted RE and MLE - Ever asthma 155

Table 5.7: Adjusted FE - Wheezing in the last 12 months 157

Table 5.8: Adjusted RE and MLE - Wheezing in the last 12 months 158

Table 5.9: Adjusted FE - Asthma medication use 160

Table 5.10: Adjusted RE and MLE - Asthma medication use 161

Table 5.11: Unadjusted marginal effects 165

Table 5.12: Fully adjusted marginal effects 165

Table 6.2: Erreygers', Concentration & Wagstaff Index: Ever asthma 187

Table 6.3: Erreygers', Concentration and Wagstaff Index: Wheezing in the last 12 months 188

Table 6.4: Erreygers', Concentration and Wagstaff Index: Asthma medication use 189

Table 6.5: Decomposition of the Erreygers' Index: Ever asthma age 3 191

Table 6.6: Decomposition of the Erreygers' Index: Ever asthma age 5 193

Table 6.7: Decomposition of the Erreygers' Index: Ever Asthma age 7 194

Table 6.8: Decomposition of the Erreygers' Index: Wheezing in the last 12 months age 3 196

Table 6.9: Decomposition of the Erreygers' Index: Wheezing in the last 12 months age 5 197

Table 6.10: Decomposition of the Erreygers' Index: Wheezing in the last 12 months age 7 199

Table 6.11: Decomposition of the Erreygers' Index: Wheezing in the last 12 months age 11 200

Table 6.12: Decomposition of the Erreygers' Index: Asthma medication use age 5 202

Table 6.13: Decomposition of the Erreyegers' Index: Asthma medication use age 7 203

Abstract

There is evidence that children from socially disadvantaged families tend to have poorer health outcomes than their richer counterparts. However, evidence on the association between family income and specific outcomes, such as asthma and wheezing, is limited. This thesis therefore applies different methodological approaches to investigate the relationship between family income and asthma-related outcomes in children, in the United Kingdom.

Chapter 1 presents the motivation and background to the thesis, and Chapter 2 describes the data and gives an overview of the methods used. Three asthma-related outcomes were used, as derived from the Millennium Cohort Study. These were: ever asthma, wheezing in the last 12 months and asthma medication use. Chapter 3 examines cross-sectionally, the association between family income and asthma-related outcomes. The results showed that income is a statistically significant driver of asthma-related outcomes, after taking into account potential confounding factors. Furthermore, for outcomes ever asthma and asthma medication use, there was some evidence that the child health-income gradient strengthened with age, novel findings in the United Kingdom.

Chapter 4 considers heterogeneity in asthma-related outcomes in children, and constructed four phenotypes based on the duration and onset of wheezing. Multinomial regression models were fitted to analyse the association between income and wheezing phenotype. Results showed statistically significant associations between income and the late-onset and persistent wheezing phenotypes, which are both likely to persist into adult life.

In Chapter 5, empirical methods are used to exploit the longitudinal nature of the data. Two approaches were used: firstly, panel data methods, where fixed and random effects models were fitted to the data. Income effects were statistically weak across all asthma-related outcomes.

Secondly, a single regression model was fitted on the combined data across all age groups for each outcome, including an interaction term between income and survey age. Results showed that higher income decreased the probability of asthma-related outcomes.

Chapter 6 examines income-related inequity of asthma-related outcomes. The first part of the analysis used Erreygers' Indices to estimate income-related inequality. Negative indices indicated income-related inequality for all ages and outcomes. Income-related inequality was then decomposed into contributing factors, and showed that income itself is the main driver.

Overall, the findings show that children from low-income families are at higher risk of adverse asthma-related outcomes, and that the burden of asthma-related disease is more concentrated in low-income children. In light of this, policies should therefore focus on reducing the degree of income-related inequality in asthma outcomes in children, by tackling the key drivers of inequality.

1.Introduction

1.1. Background

The experiences a child has in their early years provide a foundation for their whole life. In particular, health experienced in early childhood has far reaching consequences, affecting not only health and educational attainment in later childhood/adolescence, but also health, labour market participation and earnings in adult age (Case et al., 2005; Currie, 2009; Marmot, 2010; Johnson and Schoeni, 2011). Children are vital to every nation's present and future, and there is a global commitment towards children, by investing resources towards their health, development, social wellbeing and education.

The lasting impact of childhood health on educational attainment, health and social status in adulthood has been studied – among others – by Case et al (2005) using data from the National Child Development Study, an ongoing study which follows the lives of more than 17,000 people born in England, Scotland and Wales in a single week of 1958. They found strong evidence that poor parent-reported health in childhood – where health was categorised as excellent=1 to poor=4 – was strongly associated with lower educational attainment, poorer health outcomes (self-reported) and lower social status in adulthood, compared to what was achieved by individuals who had better health in childhood. Chronic conditions ranging from physical, emotional and psychological impairments that persisted before age 7 through to age 16, and those that developed between age 7 and 16, had a significant effect on health in middle age. Coefficients and standard errors (reported in parentheses) of chronic conditions in childhood regressed on adult health using ordered probit models were 0.231 (0.023) at age 23, 0.162 (0.025) at age 33 and 0.163 (0.027) at age 42, and were all statistically significant (Case et al., 2005). Furthermore, the marginal effect of family income in childhood on adult health was -0.135 at

age 33 and -0.143 at age 42, and both were statistically significant. These results support the hypothesis that income in the early years affects health in early adulthood, persisting and increasing in magnitude well into adult life (Case et al., 2005).

Johnson and Schoeni (2011) used data from the Panel Study of Income Dynamics (PSID) in the United States (US), to investigate the link between socioeconomic status and health in childhood (measured by retrospective self-reported child health) on adult health and labour market outcomes. Results showed that poor health in infancy affected adult health, resulting in a decrease of 23 points from a 100 point scale (where perfect health=100), and this was statistically significant. Their results also showed that low family income, operationalised as an income-to-needs ratio of less than one, decreased the future natural logarithm (ln) of income by 0.13, which was also statistically significant.

The combination of poor health and low income has also been shown to be an important factor in the intergenerational transmission of socioeconomic status (Currie, 2009; Johnson and Schoeni, 2011; Gondek et al., 2018). Specifically, poor health and low family income in childhood are related to lower income in adult life, less wealth, and less weeks worked. Furthermore, poor health and low income are strongly correlated to the socioeconomic status of the individual's offspring (Currie, 2009; Johnson and Schoeni, 2011). In the context of the United Kingdom (UK), a recent systematic review by Gondek (2018) synthesised evidence to assess how health at different stages of life affects economic and social outcomes in adult life, comparing these outcomes in siblings. The study concluded that children with poor physical health, chronic conditions and poor mental health have worse educational outcomes and future socioeconomic status compared to their healthier siblings.

Good health is thus seen as a precondition for children to meet their full adult potential, by maximising their overall wellbeing over the life course and allowing them to live a fulfilling life. However, not all children are afforded good health even though ill-health in childhood is

oftentimes preventable (Royal College of Paediatrics and Child Health (RCPCH), 2019). A failure to optimise the health of children may result in lower health-related quality of life and wellbeing over the life course, and a future economic burden on society that comes with a potentially unhealthy workforce (Marmot, 2010; Marmot, 2020).

1.2. Income and asthma-related outcomes

Economic literature on child health and socioeconomic status has mainly focussed on the relationship between family income and general child health (known as the child health-income gradient). This relationship has been investigated in several countries including Australia, Canada, Germany, the UK and the US, and the overall empirical evidence suggests that low family income is a significant contributor to poor health in children (Case et al., 2002; Currie et al., 2007; Currie and Stabile, 2003; Khanam et al., 2009; Propper et al., 2007; Kuehnle, 2014). A few of these studies also confirm that this relationship persists over the life of a child, leaving them to enter adult life with a potentially lower health status than their richer counterparts (Case et al., 2002). However, where the evidence for general child health is established, there are limited studies on the relationship between income and specific child health problems. In particular, the evidence on the association between family income and child asthma – the most prevalent chronic condition in UK children today – is limited.

Asthma is the most common chronic medical condition among children in the UK, with around 1.1 million children currently receiving treatment for the disease, with asthma morbidity increasing yearly (Asthma UK, 2019). Asthma, which is still a poorly diagnosed and clinically controlled condition, has large implications over many aspects of life for both afflicted children and their parents (Gosavi et al., 2016; Kavanagh et al., 2019):

- *Physical symptoms* – asthma generally causes sensitivity to the airways, which often become swollen and filled with mucus, making breathing difficult. Symptoms of asthma include chest tightness, wheezing and coughing as well as shortness of breath. These symptoms can be transient but can also be persistent once they commence. Symptoms can also vary from mild to severe and could even be life threatening (Asthma UK, 2019; Maykut et al., 2010).
- *Emotional and social wellbeing* - asthma can cause stigma in children as well as loss of self-esteem. This affects not only the child's overall wellbeing, but may cause tensions within the home, which may affect the whole family (Ritz et al., 2013).
- *Daily life and social pursuits* – asthma has been listed as the number one reason for absence from school (Ritz et al, 2013). Missed school days may in turn affect the working life of parents, who may have to take leave from work to look after their child. Practical aspects of daily life are affected, for example in avoiding asthma triggers such a dust, pollen, animal fur. Activities such as sport may also be avoided to manage asthma, and this may continue into adult life (van den Bemt et al., 2010; Ritz et al., 2013).
- *Cost to society* - The economic burden that results from asthma-related health outcomes in children is large. About 20% of the burden of asthma in the UK falls on children, yet almost 40% of asthma emergency admissions are for children below age 14 years (Mukherjee et al., 2016). Additionally, asthma in children influences missed days of work for parents who have to look after their sick children, as well as child absenteeism from school.

Given the high burden of childhood asthma, its effect on families and the society at large, it is very important to further investigate the drivers of asthma-related outcomes, in particular the role of income. This will provide a better understanding of the extent to which

low income may be a modifiable risk factor that may reduce the prevalence of asthma-related outcomes in children.

1.3. Summary of income-related policies for families with children

Given that growing up in an economically disadvantaged background negatively affects child health and development, with long-term consequences at both the individual and societal levels, it is important to understand the UK governments' strategies to tackle low income, and thus eliminate child poverty[1].

The topic of children in low-income families has become increasingly an important issue over the last two decades in the UK. In 1999, the Labour government announced a target to eradicate child poverty[2] by 2020, with interim targets set alongside it (Wickham et al., 2016). These targets aimed to lower child poverty levels to be one-quarter lower by 2004/05 and one-half of its 1999 level by 2010 (Brewer et al., 2011; Wickham et al., 2016). Policies were targeted at increasing financial support in the form of higher rates of benefits, new tax-based support and reform of child support. The government also introduced targeted programmes such as 'Sure Start', 'the Children's Funds', 'Educational Maintenance Allowances', 'Connexions' and 'On Track' (Lewis et al., 2001).

[1] Whilst in most of the economic literature, and in this thesis, economic disadvantage is operationalised by considering family income, government policy often refers to low-income settings in terms of poverty.

[2] Where poverty is defined as the proportion of individuals with household incomes less than 60% of the current median of that population

In 2010, the Child Poverty Act was passed, placing a duty on the Secretary of State to meet four child poverty targets by 2020/21 (Kennedy, 2014). It required the government[3] to produce a national Child Poverty Strategy and placed duties on local authorities and other partners to work together to achieve this goal. Policy was focussed on parental employment and skills, financial support for children and parents, information, advice and assistance to parents and promotion of parenting skills, physical and mental health, education and social services, housing, the built and natural environment and the promotion of social inclusion (Bradshaw, 2007; Brewer, 2010; Kennedy, 2014). This strategy was coupled with other government policies such as maximising employment rates, increasing the national minimum wage and investment into early years care and education (Kennedy, 2014; Child Poverty Action Group (CPAG), 2019). This saw the rate of child poverty fall substantially over the period 1999-2010, with 3.4 million children in poverty in 1999, reduced to 2.6 million in 2010 (CPAG, 2019).

In 2012, under the Conservative-Liberal Democrat coalition (2010-2015), the Universal Credit scheme was announced. From 2013, six means tested legacy benefits (which include child tax credits, housing allowance, job seekers allowance, income support, working tax credit and employment and support allowance) began to be replaced by Universal Credit. Under legacy benefits, child tax credits could be claimed for each child in a household, to aid in the cost of raising a child (excluding childcare) (Kennedy, 2012). Universal Credit on the other hand, brings together the six legacy benefits mentioned previously, is payable once monthly at the end of the month in arrears, and ensures that the total income in employment is greater than that obtained from out of work benefits, to financially incentivise people to seek employment. This is achieved by enabling beneficiaries to keep a proportion of their Universal Credit after they begin working, which is then gradually decreased as their earnings increase (Kennedy, 2012). The shift to Universal Credit is still ongoing and has been criticised, as it has been found to leave some

[3] Of England, Northern Ireland, Wales and Scotland

low-income families worse off, particularly single parent families where working in full time employment would see them worse off than working part time, and being able to claim more benefits. Therefore making the full time work an unattractive economic choice (Brewer, 2010; Kennedy, 2012; Welfare Reform and Work Act 2016; Wickham et al., 2016).

In 2014, a new government strategy was implemented to move away from supporting low-income families through financial support (in the form of benefits and tax-based support) to tackling root causes of poverty. This involved enabling parents with low-income to enter into work through job seeking services, rather than relying on income transfers from welfare. The strategy also aimed to improve the educational attainment of children from low-income families through enrolment in children centres. These strategies were aimed at improving chances of future employment, aiming to break the intergenerational cycle of low income (Wickham et al., 2016). However, in 2015 the new Conservative government removed the existing target to end child poverty by 2020, in the Welfare Reform and Work Act of 2016, generating a lot criticism and concern from opposition parties and the public (Welfare Reform and Work Act, 2016).

A recent report from the Department for Work and Pensions indicates that in 2018/2019, the prevalence of children living with relative low income remained at 30%, a figure that had steadily increased from 2010 (Francis-Devine, 2020). This indicates that policies aimed at supporting low income families with children have so far not been effective, and that more needs to be done to lift children out of poverty, increasing the likelihood of good health outcomes for them. Therefore, providing further evidence of the negative impact that low income may have on child health, specifically in prevalent conditions such as asthma, may help to strengthen the economic case for the Government to intervene and change current policy to better support low income families.

1.4. Literature review

This section discusses previous economic and social epidemiological studies that have investigated the link between low income (and other measures of socioeconomic status), and child health in the context of the UK, Europe, Australia, Canada and the US. It is subdivided into two sections, Section 1.4.1 which focusses on the association between income and measures of general child health, and Section 1.4.2. which focusses on asthma-related outcomes.

1.4.1. The association between income and child health

One of the first and highly influential studies to investigate the family income gradient in child health was conducted in the US by Case et al (2002). The authors used US based cross-sectional data from the Panel Study of Income Dynamics (PSID), the National Health Interview Survey (NHIS), the Third National Health and Nutrition and Examination Survey (NHANES) and the 1988 child health supplement to the NHIS (NHIS-CH) to examine the child health-income gradient in a sample of children ages 0 to 17 years. They found that average parental income is a statistically significant determinant of their children's health status, at all (child) ages. Both parent and physician-reported categorical measures of child health, ranging from excellent health=1 to poor health=5, were used in the analysis. Income was operationalised as the natural logarithm of average current family income. Empirical specifications were estimated using ordered probit models, which included controls for child ethnicity, child's age, family size, parent's education and marital status. Results showed that there was a statistically significant association between family income and child health, that strengthened with child age. Probit coefficients for income and standard errors (reported in parentheses) for children ages 0-2, 4-8, 9-12 were -0.16 (0.025), -0.176 (0.03), -0.202 (0.041) respectively for the parent-reported child health models, and -0.051 (0.030), -0.071 (0.036), -0.102 (0.046) respectively for the physician-

reported child health models. The income coefficients in the physician-reported child health models were notably smaller than the corresponding coefficients in the parent-reported child health models, which the authors indicate is probably due to physicians using a smaller range of values (less frequent 1 and 5 values) compared to parents, who tend to make higher and lower estimations of their child's health (more frequent 1 and 5 values) (Case et al., 2002).

Adding to the evidence provided by the Case et al (2002) study, Currie and Stabile (2003) investigated the effect of socioeconomic status, measured by permanent household income, on child health using the National Survey of Children and Youth (NLSCY) in Canada. Children were between ages 0-11 and were surveyed in 1994, 1996 and 1998. Child health was measured on a five-point scale of poor health=1 to 5 excellent health=5, which was reported by the child main caregiver. Controls for child age, family size, sex, education and whether the respondent was the birth mother, were included. Models were estimated using an ordered probit regression and coefficients for income (standard errors in parentheses) were -0.132 (0.027), -0.182 (0.020), -0.215 (0.025) for ages 0-2, 4-8, 9-12 respectively, all significant at the 5-percent level. These results were comparable to those found by Case et al (2002) in the context of the US. Interestingly, although Canadian children have universal health coverage, the child health-income gradient is still evident, and it worsens with increasing age. This has been attributed to a higher arrival rate of health shocks in children from low income families, compared to their wealthier peers, irrespective of universal health coverage (Currie and Stabile, 2003).

Using the same survey data (the NLSCY in Canada) as Currie and Stabile (2003), Allin and Stabile (2012) examined eight waves of data in children aged 1 to 15 years to investigate the effect of lagged family income on child health, as measured by a binary indicator where good/fair/poor health=1 and very good/excellent health= 0. They found statistically significant results indicating that a 1% increase in lagged family income resulted in an increase in the likelihood of reporting very good/excellent health by 0.028 in the 1-3 age group, 0.048

in the 4-7 age group, 0.052 in the 8-12 age group and 0.055 in the 13-15 age group. These findings support earlier evidence from Currie and Stabile (2003) that the child health-income gradient exists in the Canadian context and this gradient is significant and becomes stronger as children age (Allin and Stabile, 2012).

Using both cross-sectional and panel data analysis (whereas the previous two studies only used cross-sectional data), Condliffe and Link (2008) explored the relationship between family income and health in US children, using data from the Medical Expenditure Panel Survey (MEPS), 1996-2002. Child health was measured as a categorical variable where excellent health=1 and poor health=5, and family income as the average of annual income for 1996, 1998 and 2000. Results for the cross-sectional ordered probit models showed coefficients for income (and their robust standard errors in parentheses) to be -0.093 (0.018) for ages 0-3, -0.156 (0.014) for ages 4-8, and -0.175 (0.018) for ages 9-12. All results were statistically significant at the 5-percent level. In the panel data models, chronic conditions in the last five years, including asthma, epilepsy, heart problems, digestive problems and other chronic conditions, were added and results showed that children with a low family income and who had a chronic condition, had a 0.054 higher probability of being in poor health compared to those who did not. For children from families with high income, this probability was only 0.04.

Motivated by the systematic differences in the health systems in the UK (predominantly provided by the National Health System (NHS) with free care at point of delivery) and the US (predominantly provided on private healthcare insurance), Currie et al (2007) sought to investigate the relationship between family income and child health for children aged 0-15 using pooled cross-sectional data from the Health Survey for England (HSE) from 1997 to 2002 (Currie et al., 2007). Income was measured as the current pre-tax annual family income at the same time point as child health; child health was parent-reported using a 1-5 categorical scale where very good=1 and very bad=5. Using ordered probit models with controls for child age,

year, sex, ethnicity, household size, age of parents, parent's education and employment status, marginal effects[4]for income (t-statistics in parentheses) were -0.018 (10.57) for all ages, -0.027 (3.69) for ages 0-3, -0.019 (7.44) for ages 4-8, and -0.017 (6.34) for ages 9-12, and these effects were all statistically significant. Compared to the US and Canadian studies described above (Case et al., 2002 and Currie and Stabile, 2003), there is no evidence of a steepening of the gradient and the coefficient estimates are notably smaller in magnitude.

In another UK based study, Doyle et al (2007) re-examined the findings of Currie et al (2007) using the same HSE sample, and identical measures of child general health and family income. Their ordered probit models were estimated controlling for parental education, parent age, current smoking status of parents, mother smoking during pregnancy, years that a child was exposed to parental smoke, number of children in the household and year of survey. Marginal effects for family income and standard errors (in parentheses) were -0.078 (0.063) for ages 0-3 years, -0.182 (0.051) for ages 4-8, and -0.131(0.056) for ages 9-12. Results for ages 4-8 and 9-12 were statistically significant, but there was no evidence to support that the gradient increased with age. These results were similar to the findings of Currie et al (2007), who also found no evidence of a steepening income gradient with child age in the UK context (Doyle et al., 2007)

Propper et al (2007) used the Avon Longitudinal Study of Parents and Children (ALSPAC) to examine cross-sectionally the role of socioeconomic status on child health, as well as the mechanisms through which higher socioeconomic status may translate into better child health. Children up to the age of 7 years were included in the analysis. Socioeconomic status was operationalised as financial hardship, rather than family income. ALSPAC provided data on family income but this was measured less frequently than financial hardship, so financial hardship, as reported by the mother, was used in the analysis as a proxy for family income.

[4] Marginal effects here can be interpreted as the change in probability of reporting fair, bad/very bad health relative to very good or good health that is associated with a 1-percent increase in household income.

Financial hardship was based on a rating of 0-3 (3 being the highest level of hardship) indicating how difficult the mother found it to provide the following for their children: "food, heating, clothing, rent and things for the child". Child health was mother-reported and measured as a binary variable where poor health=1 and good health=0. Probit models were estimated to investigate the impact of financial hardship on child health at different child ages. Controls included were child birthweight, child sex and ethnicity, number of adults in the household, parents' education, mother's age at birth of child and parents' health. Marginal effects (and standard errors) from probit regressions at 6, 18, 30 and 81 months were 0.039 (0.012), 0.028 (0.013), 0.054 (0.013) and 0.054(0.018) respectively. All results were statistically significant at the 5-percent level, providing evidence that children from poor households experience worse health than their counterparts in better off households. Similarly to other UK based studies, there was no strong evidence to conclude that the child health-financial hardship association strengthens with age. Results from sensitivity analysis conducted using the less frequent variable family income as a measure of socioeconomic status, were comparable to those which used financial hardship.

Case et al (2008) investigated the relationship between family income and child health by re-examining the findings from Case et al (2002), that used NHIS data (1986 to 1995) from the US, and findings from Currie et al (2007) that used HSE data (1997 to 2002) from England. These two studies, respectively, showed that the child health-income gradient strengthens for US children but not for children from the UK. This could be due to the free services and equal access provided by the NHS in the UK, but it could also be due to differences in the methods, sample size, sample period and variables used. The Case et al (2008) study set out to re-examine the findings from the two studies, whilst also including additional years of data from both the HSE and NHIS up to 2005, to be able to compare across the period 1997-2005 for both data sets. They used data from the HSE including children aged 2 to 15, from the 1997-2005 HSE

studies. For the US, children from survey years 1986 to 1995 and 1998 to 2005 were included from the NHIS. The results showed that the child health-income gradient increased between ages 0 and 12 in England, in contrast to the original Currie et al (2007) study. The child health-income gradient in the US on the other hand, increased between ages 0 and 15, as in the original Case et al (2002) study. Results between England and the US were more similar in the Case et al. (2008) study, with marginal effects of income for all ages (standard errors in parentheses) from ordered probit models -0.194 (0.013) for the case of England and -0.262 (0.005) in the US, compared to -0.193 (0.015) and -0.283 (0.004) respectively, in the original Currie et al (2007) and Case et al (2002) studies. The differences in coefficients between the UK and US in the original studies could be indicative that English children have more equal access to better quality healthcare compared to US children. However, as the child health-income gradient steepens for both in this study, one could argue that the differences may in fact be due to methodological issues such as differences in wording in survey questions in the HSE and NHIS, the different sample sizes or unobservable heterogeneity.

Using the Millennium Cohort Study (MCS), Kruk et al (2013) investigated the role of income in determining health among children in the UK ages 3 to 7 years. Child health was measured subjectively as a parent-reported account of whether their children had any long-standing illness. Income was operationalised as permanent income, measured as the natural logarithm of the average of total household income when children were ages 3 to 7. Probit models were used to estimate the association between family income and the probability of having at least one long standing condition, with income coefficients (t statistics in parentheses) -0.011 (-1.63) at age 3, -0.045 (-6.05) at age 5, -0.041 (-5.25) at age 7. These results confirmed that poor health outcomes in the form of long-standing illnesses in childhood, has its roots in low family income. The findings therefore supported results by Currie et al. (2007), Propper et al. (2007) and Case et al. (2008), who all found comparable associations for UK children.

The child health-income gradient was investigated for the first time using Australian data, by Khanam et al (2009), who focused on children ages 2-7 years old using the first two waves (collected in 2004 and 2006) of the Longitudinal Study of Australian Children (LSAC). Child health was measured using a 5-point Likert scale for health where 1=excellent and 5=poor. Income was taken as the natural logarithm of the mean income of the two waves. Using ordered probit models and including controls for age and wave, sex, ethnicity, household size, parents' education and employment, housing condition, birth weight, breast feeding, they estimated an income coefficient (standard error in parentheses) of -0.029 (0.025) for ages 0-3, and -0.063 (0.027) in ages 4-8. These estimates were statistically significant and provided evidence that there exists a child health-income gradient in Australia, similar to what was found in the US in Case et al (2002) and the revised findings in the UK, shown in Case et al (2008). However, the magnitude of the association between income and child health was smaller than what was found in Canada (Currie and Stabile, 2003), the UK (Currie et al., 2007), and the US (Case et al., 2002). The smaller magnitude could also be due to lower income inequality in Australia, where the relatively low income families are not as disadvantaged when compared to the low income families in the UK, Canada and the US, allowing them to afford better housing, nutrition and assistance to help in raising children (Khanam et al., 2009; Organisation for Economic Co-operation and Development (OECD), 2019).

Using the German Interview and Examination Survey for Children and Adolescents (KiGGs), Reinhold and Jürges (2012) investigated the relationship between family income and child health in the German context. Child health was measured subjectively as a parent-reported measure on a 1-5 scale where very good=1 and very bad=5, and income was taken as the natural logarithm of current family income measured at the same time as the child health measure. Their analysis included children aged 0 to 17 years old. Ordered probit models were estimated controlling for child age, sex, household size, parity of birth, age of parents, migrant status,

parental education and employment. Marginal effects of family income (standard errors in parentheses) were -0.083 (0.016) for ages 0-3, -0.08 (0.012) for ages 4-8, -0.062 (0.011) for ages 9-12, -0.059 (0.010) for ages 13-17. Results at all ages were statistically significant, but there was no evidence of strengthening of the child health- income gradient, similar to the UK as examined by Currie et al (2007). The results showed larger coefficients than in the UK, but smaller than the US (Reinhold and Jürges, 2012).

Because of the mixed evidence in the UK found by Currie et al (2007) and Case et al (2008), surrounding the child health-income gradient, Apouey et al (2013) re-examined the child health-income gradient in children in the UK, using the Family and Children Study (FACS), a longitudinal data set which includes children from 0-17 years of age. Income was measured as the natural logarithm of average family income for all available years, and child health was parent-reported where good health=1 and 0 otherwise. Coefficients for income (robust standard errors in parentheses) from ordered probit models were 0.050 (0.035) for ages 0-3, 0.180 (0.038) for ages 4-8, 0.149 (0.041) for ages 9-12, and 0.157 (0.039) for ages 13-17. All results, except for ages 0-3, were statistically significant indicating the negative association between low income and child health. In this case, there was no evidence to support that the child health-income gradient steepens with age. These results support the Currie et al (2007) study that used HSE data (UK) for children up to age 15 and similarly found no steepening of the child health-income gradient (Apouey et al., 2013).

The most recent study of the association between family income and child health in the UK, was Kuehnle (2014) who used data from the MCS for children ages 5 and 7 – the same data set was used by Kruk et al (2013), who found that children from low income families are more likely to suffer from longstanding illness. Family income was operationalised as the logarithm of permanent income, calculated as the mean of income from the age 5 and 7 surveys. Child health was a subjective parent-reported measure taking on a value of poor health=1 to excellent

health=5. Ordinary Least Squares (OLS) models included as controls child age, sex, ethnicity, birthweight, household size, parent's age and education level and employment status, housing tenure and parental smoking. The income coefficients (robust standard errors in parentheses) were estimated to be -0.015 (0.005) for ages 4-6 and -0.009 (0.004) for ages 6-8, and were both significant at the 5-percent level. The author also instrumentalised income with exogenous labour market characteristics, using the local unemployment rate as the main instrument. The instrumental variable approach was used to account for income endogeneity that could arise from unobserved heterogeneity and potential measurement error. The results showed statistically significant results at age 5, with a coefficient of income (standard error in parentheses) of -0.128 (0.055) compared to -0.010 (0.004) in the OLS results, with no statistical significant results at ages 3 and 7 when using instrumental variables, in contrast to the OLS estimates. These results highlight the limitation of inferring causal relationship using cross-sectional analysis, the results may differ substantially when using econometric methods that can better estimate causality, such as instrumental variables or panel data methods. As with previous studies in the UK, (Currie et al., 2007, Apouey et al., 2013) the results did not show a steepening of the child health-income gradient.

The studies reviewed in this section all found a significant association between family income and child health, across the UK, Australia, Canada, Germany and the US, that low income is significantly associated with poor child health outcomes. This association was found even though health care systems (NHS versus private insurance) vary between countries. Overall, the association between income and child health was smaller in magnitude in the UK compared to the US and Canada. A steepening of the child health-income gradient was found in the US (ages 0-17) and Canada (ages 0-11). Initial research in the UK, by Currie et al (2007) (ages 0-15) did not find evidence of a steepening of this gradient, however, upon re-examining and using more data, Case et al (2008) did find evidence of a steepening gradient between ages 0 and 12.

Nonetheless findings from Doyle et al (2007), Apouey et al (2013) like Currie et al (2007), did not find evidence of a steepening of the child health-income gradient in the UK. Further research in the UK found a causal effect between income and child health by implementing an instrumental variable approach (Kuehnle, 2014), but also found no evidence of a steepening of the child health-income gradient. More research is therefore needed, to clarify these mixed results in the context of the UK.

1.4.2. The association between socioeconomic status and asthma-related outcomes

Considering specific health conditions in children, asthma is one of the most prevalent diseases of childhood in western societies, including in the UK. There has therefore been interest in understanding the drivers behind the prevalence of childhood asthma, yet the empirical evidence, especially in the health economics domain, is still limited (Propper et al., 2006; Asthma UK, 2017). This section highlights studies[5] that have investigated the association between socioeconomic status (including income) and asthma-related outcomes.

Motivated by the increasing prevalence and economic burden of asthma, Propper et al (2006) investigated whether there was an association between socioeconomic status and child respiratory health, in the UK. They used the ALSPAC data (as previously described with reference to Propper et al 2007), which included children born in 1991 and 1992 with follow-up data up to age 7 years. Socioeconomic status was measured in the main analysis by father's occupational status at time of survey, categorised in five classes ranging from professional (I) to unskilled (V), and by current family income when children were aged 3 and 4, in a sensitivity analysis (Propper et al., 2006). Child respiratory health was parent-reported at ages 6, 18, 30, 42

[5] These studies are detailed in Appendix A-6.

and 81 months. Child respiratory health outcomes were operationalised as dichotomous measures=1 for: asthma at 81 months (whether a mother reported her child having asthma at 81 months), ever wheezed (a child ever having wheezing at any of the five time points mentioned previously), and two measures of persistent wheeze (wheezing reported at least three out of the four observations taken between birth and 42 months; or the latter plus wheezing or asthma at 81 months). Logistic regressions were estimated controlling for a number of factors including birthweight, exposure to other children in infancy (number of siblings at 8 months and day care attendance), child's diet, mother's age, maternal smoking, parental history of asthma, housing conditions, and local deprivation. Results showed that the odds ratios (ORs) and standard error in parentheses, for occupational class and respiratory health outcomes were 1.06 (0.03) for asthma at 81 months; 0.98 (0.02) for ever wheezed, 1.03 (0.04) for persistent wheeze from birth to 42 months, and 1.14 (0.06) for persistent wheeze from birth to 42 months and asthma/wheezing at 81months (Propper et al., 2006). Only the latter category of persistent wheeze showed a statistically significant association. In the sensitivity analysis, in which socioeconomic status was proxied by family income, results were statistically significant for asthma at 81 months, where increased income was associated with a decreased likelihood of asthma, and the odds ratio (standard error in parentheses) was 0.80 (0.060) for fully adjusted models (Propper et al., 2006). Results from the both the base case and sensitivity analyses showed that there is a statistically significant association between socioeconomic status and poor respiratory health.

Using the data from the UK based MCS, Taylor-Robinson et al (2015) classified wheezing by longitudinal pattern into phenotypes/categories as informed by Martinez et al (1995). They investigated the association between socioeconomic status, measured by mother's highest level of education (based on National Vocational Qualifications (NVQ) or equivalent groups) and wheezing patterns in children at ages 3, 5 and 7. Wheezing was categorised into three possible

phenotypes, based on onset and time course: early remitting, persistent wheezing, relapsing wheeze and transient/intermittent wheezing with never wheezed as the reference group. A sample of 11,141 children was used in the analysis. Unadjusted results showed significant relationships between maternal education and wheezing, where children of mothers who had no educational qualifications were approximately 40% more likely to have transient wheezing in early years, and about 30% more likely to have persistent wheezing (up to age 7) (Taylor-Robinson et al., 2015). This study highlighted that once maternal age, maternal smoking and breastfeeding are controlled for, the socioeconomic differences in the prevalence of wheezing phenotypes disappear.

Violato et al (2009) used MCS data (children age 9 months, 3 and 5 years old) to investigate the relationship between household income and child respiratory health in the UK, using both cross-sectional and panel data methods. For the cross-sectional analysis, family income was measured using both transitory lagged income and permanent income (both in their natural logarithmic form), and respiratory health was measured using two dichotomous measures of asthma and wheezing at age 3 and age 5, with a combined measure of asthma and wheezing at 9 months (Violato et al., 2009). In the unadjusted models, partial effects and standard errors derived from estimated probit models were 0.014 (0.005) at 9 months for asthma/wheezing, -0.041 (0.006) and -0.040 (0.009) at age 3, and -0.043 (0.008) and -0.040 (0.011) at age 5, for asthma and wheezing outcomes respectively. Estimates were statistically significant at age 3 and 5 for both asthma and wheezing, but not at 9 months. In models adjusted for other measures of socioeconomic status, child characteristics, parental health risk factors, mother's child-health related behaviour and environmental exposure, income partial effects were 0.005 (0.006) at 9 months, -0.014 (0.008) and -0.012 (0.013) at age 3; -0.014 (0.008) and -0.025 (0.014) at age 5 for asthma and wheezing respectively. All the associations were statistically significant (except for wheeze at age 3 and at 9 months), although to a lesser extent than the unadjusted models. The

fixed effects model, exploiting the panel dimension of that MCS data, showed a weak association between income and child respiratory health, with a coefficient and p-value in parentheses of -0.012 (0.13). These results indicate that after controlling for the above-mentioned variables, there still exists an association between income and child respiratory health. However, as with general child health in the context of UK, there is not enough evidence to support a steepening of the child health-income gradient.

Using the longitudinal Dutch Generation R study, Hafkamp-de-Groen (2012) investigated the association between socioeconomic factors and wheezing and breathlessness. Socioeconomic status was measured by level of family income, categorised into three levels <€2000, €2000-3000, and >€3000 per month. Respiratory symptoms were measured when children were aged 1, 2, 3 and 4 from parent-reported accounts of wheezing and breathlessness in the last year. Results showed that at ages 3 and 4, children from low income families were at higher risk of wheezing compared to high income children, with adjusted odds ratios (aOR) equal to: 1.57 (95% CI:1.09-2.26) and 1.53 (95% CI:1.06-2.22), respectively.

Using the same Generation R study described above, Hafkamp-de-Groen 2013 examined the association between socioeconomic status, measured by net family income when children were aged 2, and its influence on wheezing and asthma in the same children aged 6. It was found that children from families with low income (<€2000 a month) were at increased risk of wheezing (but not asthma) compared to children from families with high income (>€2000 a month), with an aORs equal to 1.43 (95% CI:1.10-1.88), where >€2000 is the reference point.

The Kuehnle (2014) study, which found a statistically significant causal relationship between family income and child health in the UK, also extended their methodology to include investigating the association between family income and specific long-term conditions, including respiratory health, using the MCS data. The OLS estimate for income regressed on long term respiratory health (standard error in parentheses) in the fully adjusted model was -

0.013 (0.004) and was statistically significant at the one percent level. The results showed that low family income is statistically associated with a decrease in the probability of having a respiratory-related long-term condition, such as asthma and wheezing (Kuehnle, 2014).

The literature on income and child asthma-related outcomes in the health economics context is limited. Yet, the relationship is important to examine as asthma remains the most prevalent chronic condition in UK children. As with general child health, asthma-related outcomes are likely to also be partly determined by family income. Studies so far focus mainly on asthma-related outcomes in early childhood, hence there is a need to include data from later childhood to be able to paint a clearer picture of this association between family income and child asthma-related outcomes, and how it may change with age.

1.5. How income determines child health outcomes

From the literature on the determinants of child health outcomes and asthma-related outcomes, there is strong evidence to support that there is a statistically significant association with family income. Even after adjusting for potential confounding variables, there remains evidence to indicate that income has a direct association on child health outcomes, with low income being associated with adverse health outcomes. This association may be explained by a) transmission mechanisms b) a direct association in which low income influences child health outcomes, even after taking into account possible transmission mechanisms. It is important to distinguish between these two elements, as the association between income and child health is often explained by a combination of the two, due to the complex and intercorrelated pathways between socioeconomic status and child health (Propper et al., 2006). The following section expands on these two points.

1.5.1. Transmission mechanisms

Studies investigating the association between socioeconomic status/family income and child health outcomes, including child asthma, usually report both unadjusted and adjusted estimates. Fully adjusted economic models often include the two following categories of explanatory variables or confounders, namely a-priori variables and potential mediators. A-priori variables are factors that existed at birth and factors whose outcomes are unchangeable such as child age and ethnicity.

The second category are explanatory variables which function as potential transmission mechanisms, when they may lay in the causal path between socioeconomic status/family income and child health outcomes. Transmission mechanisms essentially act as a channel through which income may influence child health and there are two widely used explanations for this mechanism. Firstly, the 'investment theory' proposed by Becker (1986) which states that outcomes of children depend on biological and cultural traits that are automatically transmitted from parents to children (Becker, 1986). The theory stipulates that the intergenerational mobility of earnings depends on the inheritability of endowments, which in turn depend on the ability of parents to invest in goods and services for their children, such as good quality housing, leisure time, extracurricular activities, a healthy diet, sports clubs. Essentially, factors that are known to directly contribute to good child health outcomes, in this case better asthma-related outcomes. The theory assumes utility maximisation by parents who are concerned about the welfare of their children.

Secondly, the 'good parent theory' in which low income affects parents' ability to be 'good' parents, i.e. the financial stress reducing their non-monetary investment in their children such as a reduction in the quality of time spent with them (Mayer, 2002). The theory suggests that low income is associated with symptoms of stress, and that symptoms of stress in turn are

associated with poor parenting practice. Researchers have agreed that poor parenting may cause adverse health outcomes in children. In the specific case of asthma-related outcomes, this may mean that mothers with poor mental health may be less likely to adhere to asthma treatment regimens, less consistent in the quality of care they give their children and, potentially less likely to seek medical care for ongoing chronic conditions in children. Transmission mechanisms can therefore be included in analysis by introducing explanatory variables.

Although there is no clear distinction between the grouping of explanatory variables in the literature looking at socioeconomic status/family income and its impact on child health outcomes, a few categories of factors have been defined for the purposes of this thesis, as informed by the relevant economic and social epidemiological literature reviewed (these are described further in Chapter 2.10.):

- Measures of socioeconomic status (other than family income). These include parental education, mother's age at birth of child, which may pick up issues that explain poor child health. For example, parents with a low level of education may be unable to manage chronic conditions in children. Likewise, low maternal age at child birth may have less experience at adapting good parenting practices and management of their child's health.
- Parent and child health-related behaviours, which includes factors such as smoking during pregnancy and alcohol consumption during pregnancy, both which may lead to an unhealthy uterine environment leading to low birthweight, which may then predispose a child to diseases such as asthma. This category also includes factors such as a parent's mental and physical health, which both may affect child health outcomes by influencing a parent's ability to care for their child in general and manage child health conditions. Factors such as breastfeeding duration are also included in this category of variables. Breastfeeding has been widely documented to be associated with child health

outcomes through the protective components in mother's milk, a lack of which may lead to conditions such as asthma in the early years (Quigley et al, 2018).

- Environmental factors such as exposure to tobacco smoke, pollutants, dust, and allergens such as pollen, household heating, damp or condensation in the home, which may act as direct triggers to childhood health outcomes including asthma and wheezing.

1.5.2. The direct effect of income

There are mixed findings on whether income itself has a direct association with child health outcomes (Case et al., 2002; Currie et al., 2007; Propper et al., 2006; Propper et al., 2007). A recent systematic review by Cooper and Stewart (2020) succinctly summarises findings from studies that have investigated the association between income and child health outcomes. It supports the notion that money in itself does matter for children's health outcomes, even once confounding factors are taken into consideration (Cooper and Stewart, 2020). What this means is that poorer children have worse outcomes *because* they are poor, and this is not completely attributed to other determinant factors that are also associated with income such as other socioeconomic factors, parent-child health related behaviours and environmental factors. Evidence has shown that whilst an increase in household income may not completely eliminate disparities in health outcomes between low-income and high-income children, it can contribute to a substantial reduction in such disparities (Cooper and Stewart, 2020).

1.6. Changes in the child health-income gradient

Secondary to exploring whether an association between family income and child health exists, a few studies have also investigated how this association may strengthen with child age (Case et al., 2002; Currie and Stabile, 2013; Currie et al., 2007; Propper et al., 2006; Chen et al., 2006, Apouey et al., 2013; Kuehnle, 2014). Any patterns of change in the gradient may highlight the

importance of socioeconomic status throughout childhood, rather than at a single point in time. Evidence of a strengthening of the gradient has previously been explained by two theories. One explains that poor children have more health shocks as they age, compared to their richer counterparts, making the importance of income more crucial throughout childhood (Currie and Stabile, 2003). The other suggests that the strengthening of the income gradient with child age, could be indicative of the fact that adverse health outcomes experienced in early childhood have long lasting effects, which have accumulated over time (Currie and Stabile, 2003). In my thesis, I aim to contribute to this issue of the child health-income gradient by providing new empirical evidence, in relation to asthma-related outcomes. Such evidence could be used to inform policies aimed at improving the living standard of low income children from the very early years, and potentially prevent worse health outcomes later on in childhood.

1.7. Thesis objectives

Low income has been found to be significantly associated with poor health outcomes in children across the globe. The rise of the prevalence of childhood asthma, and associated outcomes such as wheezing, makes it a highly pressing health issue. In addition, there is also a very high number of children living in low income families in the UK (30% living in poverty, as defined by the OECD[6]) especially compared to other OECD countries. Health in childhood is crucial for determining outcomes in adulthood, in terms of both health and socioeconomic status, hence investigating determinants of widespread chronic child health conditions like asthma, may shed light on what factors need to be addressed by governments and the wider society to ultimately improve asthma-related outcomes in children.

[6] People are classified as poor when their equivalised disposable household income is less than 50% of the median prevailing in that country (OECD)

This thesis aims to provide answers to the gaps in the literature by making use of a longer follow-up period for children in the UK context, including longitudinal data on asthma-related conditions and family income, collected over 11 years in the MCS. Whereas most studies have used only cross-sectional data to explore the association between family income and asthma-related outcomes in children, this thesis will in addition to cross-sectional analysis, use panel data methods to explore if there are both time-varying and time-invariant determinants of asthma-related outcomes (in addition to income itself which is time varying), made possible by the existence of a rich panel dataset.

It will also explore more complex measures of asthma-related outcomes beyond binary outcomes and examine patterns of wheezing or wheezing phenotypes to see if family income may be associated with specific patterns of childhood wheezing based on onset and duration. This is a novel approach in terms of combing wheezing phenotypes over an 11year follow-up period, but also a novelty in the field of health economics to the best of my knowledge

In addition to looking at the association between family income and asthma-related outcomes, this thesis aims to use a measure of income-related health inequality to examine whether there is any income-related inequality in asthma-related outcomes. Income-related inequality in different health outcomes is an issue in the UK and other high income countries, yet there are very few studies which focus on inequality in specific health outcomes (Walsh and Cullinan, 2015).

The research objectives of this thesis are outlined below:

1. To examine, using cross-sectional analysis, whether there is an association between family income and asthma-related outcomes in children at age 3, 5, 7 and 11 and to examine if this association changes with age (a child health-income gradient) (Chapter 3);

2. To investigate the extent to which the association between family income and child asthma can be explained by a priori variables (prenatal, demographic, hereditary) and other potential explanatory variables, some of which are possible transmission mechanisms (socioeconomic factors, mother and child health-related behaviours, and environmental and household factors) (Chapter 3);
3. To select appropriate wheezing phenotypes and to investigate the association between family income and these phenotypes. The role of a priori and other potential explanatory factors will also be investigated (Chapter 4);
4. To explore, using panel data methods - which consider changes in both family income and health outcomes over time - the association between family income and asthma-related outcomes. The role of a priori and other potential explanatory factors will also be investigated (Chapter 5); and finally,
5. To measure income-related health inequality in asthma-related outcomes and subsequently decompose the inequality into contributing factors (Chapter 6).

2.Methods

2.1. The Millennium Cohort Study

This thesis uses data from the Millennium Cohort Study (MCS) to investigate all research objectives described at the end of Chapter 1. The MCS is a multidisciplinary ongoing longitudinal study of 18,818 children from 18,552 families, who were born throughout the UK between September 2000 and January 2002. It is the youngest of the UK's well renowned series of national cohort studies which include the National Survey of Health and Development (NSHD), the National Child Development Study (NCDS) and the British Cohort Study (BCS) The MCS is managed at the Centre for Longitudinal Studies (CLS), based at the Institute of Education at University College London and is funded by the Economic and Social Research Council (ESRC) as well as other agencies, including the UK government.

The study cohort is specifically drawn from children born between 1 September 2000 and 31 August 2001 (for England and Wales) and between 24 November 2000 and 11 January 2002 (for Scotland and Northern Ireland) who were alive and living in the UK at age 9 months, and eligible to receive child benefit (Connelly and Platt, 2014). Only a small group of children in the UK were ineligible for child benefits – mainly asylum seekers. The MCS is distinct from previous British cohort studies in that it includes babies born over a full year, rather than at a specific time of the year. The sample was constructed to be representative of the whole UK population, and to ensure this, the MCS oversampled children from deprived backgrounds as well as children from areas of relatively high ethnic minority concentration. Oversampling in this way allowed typically hard to reach populations to be accurately represented (Connelly and Platt, 2014).

The MCS provides information on a wide range of experiences and outcomes of children and their parents. It includes data on child and parental health, birth and infancy data such as delivery mode, birthweight and breastfeeding, parental health-related behaviours such as alcohol consumption and smoking, parental employment, income, education and housing. Each survey contains a number of modules completed by each respondent (usually mother or father) (Section 2.4). Surveys were conducted when children were age 9 months (MCS 1) and age 3 (MCS 2), age 5 (MCS 3), age 7 (MCS 4), and age 11 (MCS 5)[7].

2.2. Sampling in the MCS

The MCS includes children born in all four countries of the UK: England, Wales, Scotland and Northern Ireland. The population is stratified by country and within each country - by area of residence (referred to as electoral wards) according to whether the wards have at least 30% ethnic minority population, whether socially disadvantaged (poorest 25% of wards in terms of the Child Poverty Index (CPI)) or advantaged (not falling into the top quartile of the CPI) (Plewis and Kallis, 2008). Wards in England are classified into three strata: ethnic minority, disadvantaged, and advantaged stratum. Wales, Scotland and Northern Ireland due to a low prevalence of ethnic minorities, only have two strata each: disadvantaged and advantaged. The MCS variable used to identify stratum is *pttype2* and the clustering variable, which is the primary sampling unit is electoral ward, *sptn00* (Hansen, 2014).

2.3. Survey protocol

Prior to the fieldwork for MCS 1, the Department for Work and Pensions (DWP) sent opt out letters to all parents who had a child with an eligible birthdate, and registered as living in one of

[7] The age 14 (MCS 6) and age 17 years (MCS 7) have been released since writing this thesis.

the sample electoral wards. Letters including information leaflets were sent in batches every 4 weeks to families whose babies were about 7 months old (Hansen, 2014). Parents who opted-out were removed from the sample. Fully trained interviewees conducted interviews with consenting families in 17 consecutive waves across England, Wales, Northern Ireland and Scotland, with the aim to have as little overlap between waves as possible. Interviewers arranged interviews as soon as possible after addresses of families were issued and interviews were carried out between June 2001 and January 2003. Fieldwork was also conducted in 17 waves for MCS 2 between September 2003 and January 2005. MCS 3 was driven mainly by the requirement for the family to be interviewed during the child's first year of schooling. Fieldwork was carried out in two waves for each country during the period January 2006 to December 2006 for the main survey. Similarly, MCS 4 and 5 were driven by the requirement for the child to be in their third year and final year of primary schooling, respectively. The main surveys for MCS 4 were carried out from January to December 2008 and January 2012 to February 2013 for MCS 5 (Hansen, 2014).

2.4. Survey content

Each of the five surveys MCS 1-5 contained a number of elements which varied by content, mode of survey (interview, self-completion or observation) and respondent. An excerpt of survey elements from MCS 1 and MCS 2[8] as used in this thesis is provided in Table 2.1.

Table 2.1: Survey elements MCS1 and MCS2

Respondent	**Mode of interview**	**Summary of Content**
Mother/Father	Face to face interview	Household module (details of respondent relationship to child, country, household composition, language)
Mother/Main	Face to face interview	Module C: Pregnancy, labour, delivery

[8] Summaries for survey elements contained in subsequent cohorts can be found in Appendix A-2.

		Module D: Baby's health and development
		Module G: Parental health
		Module J: Employment, income, education
		Module K: Housing and local area
Father/Partner	Face to face interview	Module G: Parental health Module J: Employment and education

2.5. Structure of the MCS datasets

Data collected at each MCS survey were made available through multiple computer files – at the individual, family and geographical level – and these needed to be linked both cross-sectionally and longitudinally in order to be able to use them for the analysis. For each of the five MCS surveys, the data files included were (official study names given in parentheses):

- A longitudinal file ("MCS longitudinal family level information"), which includes information on the cohort member's family. This file was updated at every survey as necessary, for example, when the family structure changed, and new family members were added or removed. From this I extracted the unique identifying number of the cohort member, stratum, population correction factor and weighting variable.
- The main cross-sectional dataset ("Parent Interview Data") which includes family and individual-level data for cohort members and parents. From this I extracted information on child and parent health related variables such as delivery mode, breast feeding, mother's general, mother's mental health, smoking during pregnancy, alcohol consumption during pregnancy, child health and household environmental factors.

- Cross-sectional household grid data ("Household Grid") for MCS 1-5, which includes one row for each person in the household grid. This data was used to enable linkage by cohort member, for example, to the geographical data described below.
- Geographically linked data ("Geographically linked data") for MCS 1-5 from which I extracted data on rural-urban morphology.
- Derived variables ("Derived variables" for MCS 1-4 and "Family Level Derived Variables" for MCS 5). I extracted data on income, mother's BMI before birth of child, birthweight, number of siblings, mother's highest level of education, age of mother at birth of cohort child and housing tenure.

2.6. Response rate

The MCS aims to track all the original 18,552 families who took part in MCS 1. The productive families[9] included in Table 2.2 take into account all families who were present in MCS 1, except for ineligible families (emigrant families and deaths), families with uncertain eligibility (those that were temporarily away from the UK, and untraced movers) and unproductive families (refusals, non-contacts, other non-responses including language problems, ill/incapacitated, deleted/lost data).

Survey weights provided in the MCS datasets were used to account for the disproportionate representation of areas with high proportions of ethnic minorities in England, residents living in areas with high levels of child poverty as well as residents in Northern Ireland, Scotland and Wales. Survey weights also include an adjustment for attrition to account for possible biases

[9] Productive families are those with some data from at least one of the six data collection instruments: main interview, partner interview, proxy partner interview, Benchmark Assessment System (BAS), Bracken School Readiness Assessment, height and weight.

generated by systematic non-response. The survey weights used for this thesis were one overall weight incorporating sample weights and non-responses weights[10] (Hansen, 2014).

Table 2.2: A summary of study population and response in the MCS surveys

Survey	**MCS 1**	**MCS 2**	**MCS 3**	**MCS 4**	**MCS 5**
Year data collected	2001/2	2003/4	2005/6	2008/9	2012/2013
Age of children	9 months	3 years	5 years	7 years	11 years
Productive families (n) (includes families with singletons, twins and triplets)	18,552	15,590	15,246	13,859	13,287
Productive families out of all families eligible for inclusion at the start of each survey (%)		80.0	79.2	72.0	69.1

2.7. Study population

Inclusion criteria to define the study population that was used throughout this thesis are:

- The cohort member is a singleton child;
- The main respondent is the birth mother of the cohort member;
- The cohort child is successfully tracked in all of the five MCS surveys.

The 246 sets of twins and 10 sets of triplets recorded at MCS 1 are excluded because these numbers are too small to analyse separately and detect significant associations with asthma-related outcomes. Considering only families where the main respondent is the birth mother allowed me to fully exploit information on pregnancy and birth-related factors such as smoking and alcohol in pregnancy, mode of birth, birth weight and breastfeeding duration. Including

[10] Details on the calculation of weights can be found in Appendix A-3.

only cohort members present in all five surveys allowed the same group of children to be analysed, and therefore provided results that can be more reliably compared over time, for example, attributing observed effects to changes over time rather than to variability between different samples of children.

Because of these inclusion criteria, the maximum sample size in all of my empirical chapters is 10,260 children/families (Figure 2.1). Within each chapter, however, the actual number of children/families included in each empirical model varies due to availability of data on asthma-related outcomes, family income and other explanatory variables across surveys. This variation in sample size is due to the following points:

- Only children present in all five MCS surveys with data available for all relevant variables are included in each analysis[11]. That is, missing data are handled using a listwise deletion approach. I investigated previous literature that used the MCS data and imputed missing data, with results showing that there were no differences in estimations when imputed data was used compared to data where listwise deletion was implemented (Violato et al., 2009). This provided evidence that using a listwise approach in this thesis was suitable. Additionally, there is relatively little missing data due to item non-response (<0.3% for all asthma-related outcomes and around 2% for family income, at all ages) and therefore a complete case analysis is suitable;
- Missing data due to survey refusal non-response (which ranged between 9.4% at MCS 2 to 22.9% at survey 5) and missing data due to attrition is addressed using non-response

[11] A complete list of all variables used in the analysis can be found in Appendix A-1.

weights which take both of these items into account, these were calculated by the CLS (Hansen, 2014).

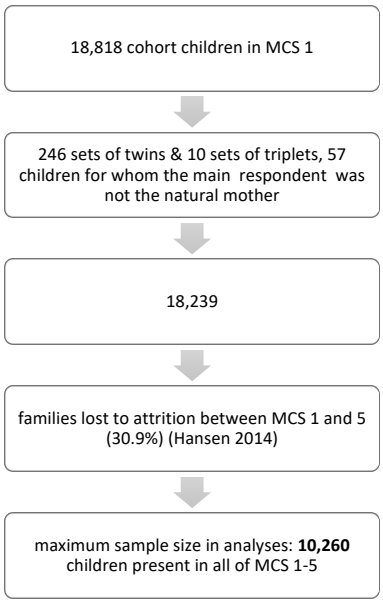


Figure 2.1: MCS research population

2.8. Asthma-related outcomes

Three dichotomous (with value of 1 if yes and 0 if no) asthma-related outcomes were used in this thesis: 1) ever asthma 2) wheezing in the last 12 months and 3) asthma medication use. These outcomes were derived from different questions asked of the main respondent at MCS 2 to 5[12] (Table 2.3).

Table 2.3: MCS 2-5 asthma-related outcomes

MCS question	**Response options**	**Asthma-related outcome**	**Surveys present**
Has ^Jack ever had asthma?	Yes No Don't know	Ever had asthma	Age 3, 5,7, 11
If ^Jack has a wheezing or whistling in the chest in the past, if yes then, Has ^Jack had	Yes No	Wheezing in the last 12 months	Age 3, 5, 7, 11

[12] Asthma-related outcomes derived from MCS questions are parent-reported measures hence are subjective and may be subject to reporting bias.

MCS question	Response options	Asthma-related outcome	Surveys present
wheezing in the chest in the last 12 months?			
Is [^Cohort child's name] currently taking any medicines on a regular basis that were prescribed by a doctor or hospital? By regular I mean every day for two weeks or more	Yes No		Age 5, 7, 11
If cohort child taking medicines regularly, please could you tell me the name of the medicine (repeat for up to six medicines)[13]		Asthma medication use	Age 5, 7, 11

These questions are all validated questions taken from the International Study for Asthma and Allergies in Childhood (ISAAC), an international epidemiological research study that was established in 1991 to study asthma, eczema and rhinitis in children. Each of the outcomes in Table 2.3 were operationalised in the empirical analyses[14] as binary variables. In the first survey (MCS 1) questions pertaining to asthma-related outcomes were worded differently to subsequent surveys, as asthma is not usually diagnosed in young infants, for this reason asthma data from MCS 1 were excluded. Table 2.4 outlines the questions from which asthma-related outcomes were derived in MCS 1. In MCS 2-5, a questionnaire on child health (Module D) was completed by the main respondent. These questions are displayed in the first three rows of Table 2.4.

Table 2.4: MCS 1 asthma-related outcomes

MCS question	Response option	Survey age
We would like to know about any health problems for which ^Jack has been taken to the GP, Health Centre or Health visitor, or to Casualty, or you have called NHS direct.	Yes No	9 months

[13] The respondent is then directed to consult a code book to identify drugs which included Alupent, Atrovent, Beclazone, Becloforte, Beclometasone dipropionate, Becotide, Bricanyl, Flixotide, Phyllocontin continus, Pulmicort, Pulmicort turboinhaler, Pulmicort respules, Salamol, Salbutamol, Salmeterol, Serevent, Tilade Mint, Uniphyllin Continus, Ventodisks, Ventolin, Volmax.

[14] This excludes Chapter 4 where the binary response to wheezing in the last 12 months was used to distinguish between patterns of wheezing.

If there were any problems, what was this problem? (select up to 8 health problems) option 5: wheezing or asthma		

2.9. Family income

The main exposure in the analysis is family income. Family income is a measure of socioeconomic status, widely used in economics literature, that captures a family's ability to purchase desired material resources and also provides information on access to social and economic resources (Galobardes, 2006). It measures a wide range of circumstances that may have direct implications for health - a parent's ability to access better quality resources such as food, shelter, leisure activities, or child care, that may all be determined by family income.

In MCS 1-5, main respondents were presented the card in Figure 2.2 and asked the following question:

"*Which of the groups on this card represents your (and your husband's/wife's/partner's) total take-home income from earnings, benefits and any other sources of regular income, after tax and deductions. Please include council tax benefits and housing benefit even if they are paid directly to your landlord. Just tell me the number beside the row that applies to you*".

Respondents could select one response from a range of income bands given in weekly, monthly and annual amounts. Where respondents did not know, refused to answer or responded, "not applicable", these data were coded as missing. Pseudo-continuous income values were created based on the list of income labels for both two parent households and lone parent households using interval regression conducted by the CLS team. This method allowed for a continuous value of income within each income band to be computed rather than assuming all cases within a band take the midpoint income value. These income values were equivalised[15] using OECD

[15] See Appendix A-4 for details of equivalisation.

scales. At age 11, collection of income data was enhanced to probe respondents on other sources of take home pay other than their main income source.

	WEEKLY	MONTHLY	ANNUAL	
2	**Less than £30**	**Less than £130**	**Less than £1,600**	2
3	**£30 less than £60**	**£130 less than £260**	**£1,600 less £3,100**	3
4	**£60 less than £90**	**£260 less than £390**	**£3,100 less than £4,700**	4
5	**£90 less than £120**	**£390 less than £520**	**£4,700 less than £6,200**	5
6	**£120 less than £150**	**£520 less than £650**	**£6,200 less than £7,800**	6
7	**£150 less than £200**	**£650 less than £870**	**£7,800 less than £10,400**	7
8	**£200 less than £250**	**£870 less than £1,100**	**£10,400 less than £13,000**	8
9	**£250 less than £300**	**£1,100 less than £1,300**	**£13,000 less than £15,600**	9
10	**£300 less than £350**	**£1,300 less than £1,500**	**£15,600 less than £18,200**	10
11	**£350 less than £400**	**£1,500 less than £1,700**	**£18,200 less than £20,800**	11
12	**£400 less than £500**	**£1,700 less than £2,200**	**£20,800 less than £26,000**	12
13	**£500 less than £600**	**£2,200 less than £2,600**	**£26,000 less than £31,200**	13
14	**£600 less than £700**	**£2,600 less than £3,000**	**£31,200 less than £36,400**	14
15	**£700 less than £800**	**£3,000 less than £3,500**	**£36,400 less than £41,600**	15
16	**£800 less than £900**	**£3,500 less than £3,900**	**£41,600 less than £46,800**	16
17	**£900 less than £1,000**	**£3,900 less than £4,300**	**£46,800 less than £52,000**	17
18	**£1,000 less than £1,550**	**£4,300 less than £6,700**	**£52,000 less than £80,000**	18
19	**£1,550 or more**	**£6,700 or more**	**£80,000 or more**	19

Figure 2.2: Card from the MCS Computer Assisted Personal Interview (CAPI) questionnaire showing weekly, monthly and annual income bands, after tax

2.9.1. Income for thesis data analysis

2.9.1.1. Permanent income

In the base case empirical analysis of Chapter 3 (Cross-sectional analysis), Chapter 4 (Income and wheezing phenotypes) and Chapter 6 (Income-related inequality in asthma-related outcomes), family income was operationalised as permanent income, which assumes the economic theory that individuals usually spend money according to their long-term average income, rather than their current or transitory income (Case et al., 2002; Friedman, 2008). This theory has been supported by previous literature that has found a larger association between income and child health when measured considering permanent income, compared to transitory income (Berger et al., 2009). In this thesis, permanent income was calculated from the annual

OECD equivalised family income, which is available in the derived variables dataset for each MCS survey. The following steps outline the permanent income calculation:

1. I identified the annual OECD equivalised family income for each survey (where income is reported as mean weekly or monthly income. I multiplied these figures by 52 and 12 respectively to obtain annual income);
2. Annual income figures from step 1 were inflated to 2011 prices (the year in which the latest survey included in my analysis) using the Average Weekly Earnings (AWE)[16] index;
3. Permanent income was then calculated as the mean annual family income, adjusted for inflation in step 2, up to and including the age at which the asthma-related outcome was measured. For example, at age 3, permanent income is the mean of OECD equivalised and inflated income at 9 months and age 3; at the age 5, it was the mean of OECD equivalised and inflated income at 9 months and ages 3 and 5; at the age 7, it was the mean of OECD equivalised and inflated income at 9 months and ages 3, 5 and 7; and at the age 11, it was the mean of OECD equivalised and inflated income at 9 months and ages 3, 5, 7 and 11 years;
4. The natural log of income was used as it is commonly assumed that the relationship between family income and various health outcomes is non-linear in child health literature (Case et al., 2002; Currie and Stabile., 2003; Case et al., 2005; Currie et al., 2007).

[16] The AWE index was officially introduced by the Office for National Statistics in 2009. Historical data were available which allowed the inflation rate to be calculated prior to 2009. See Appendix A-5

2.9.1.2. Transitory income

Transitory income was used in sensitivity analysis (in Chapter 3) as an alternative measure of family income. Using transitory income also allows one to check the robustness of the base case results, to assess whether long-term income or episodic income more strongly determines child health outcomes. In this thesis, transitory income was measured as lagged transitory income. This was equivalent to OECD equivalised family income in the survey year preceding the time period in which the asthma-related outcome is measured, expressed in 2011 prices. For example, in the models at age 3, lagged transitory income was measured at age 9 months; at age 5, lagged transitory income was measured at age 3, and so on. Using lagged transitory income rather than addresses problems of reverse causality that might occur with the use of current transitory income, which itself may be influenced by parental health (Case et al., 2002; Currie et al., 2007; Violato et al., 2009; Kuehnle 2014).

2.10. Other explanatory variables

While income is the main exposure in the analysis, there are other factors that have been found in the literature to be consistently associated with asthma-related outcomes, and these were explored in the statistical models. This section describes such factors which are grouped into four categories, firstly:

- A priori variables

Then categories of other potential explanatory variables:

- Other measures of socioeconomic status
- Mother and child health-related variables
- Household and environmental factors

2.10.1. A priori variables

These include perinatal factors, demographics and hereditary factors that are considered largely, as unchangeable. These factors are generally time-invariant and are controlled for in all of the analyses in this thesis.

- **Ethnicity**

The literature shows that there are large variations in the risk of asthma between ethnic groups. Specific ethnicities have been found to have an elevated risk of asthma and wheezing in childhood, with higher prevalence being found in black, Pakistani and Bangladeshi children compared to white children (Sheikh et al., 2016). In the MCS, respondents are asked: "*And which of the groups on this card do you regard [^Cohort child's name or twins] as belonging to?*". Cohort members are grouped into one of the following categories: White, black African, black Caribbean, Bangladeshi, Indian, Pakistani, Chinese, mixed or other. Ethnicity is then coded as a categorical variable with nine categories with white children being the reference group.

- **Age**

The exact age of children varies within each MCS survey due to data collection being conducted over 12 months rather than at one exact time point. The spread of age at a given survey in the MCS is not large, but age is included because changes in child health and development can happen over just a few months. Age is measured in months as a continuous numeric variable.

- **Sex**

An increased risk of asthma in boys before puberty has been found in several studies (Propper et al., 2007; Arathimos et al., 2017). Sex is also a commonly included confounder across the literature in many fields pertaining to health and economics and epidemiology and differences

are commonly found according to sex. Therefore, child sex is included in the statistical analysis to control for potential differences due to sex. In this thesis, sex is measured as a dichotomous variable coded as 1 for males and 0 for females, with females representing the reference group.

- **Birthweight**

Previous studies have identified a strong association between low birthweight and asthma-related outcomes with the effects of low birthweight on lung function and respiratory health being most pronounced in early childhood (Boardman et al., 2002). It has also long been identified that low birthweight is strongly associated with wheezing in early childhood (Sears et al., 2003). In the MCS, respondents were asked their child's weight at time of birth. For the statistical analysis, birthweight was operationalised in three categories according to the World Health Organisation (WHO) classification: very low birthweight (less than 1.5kg), low birthweight (between 1.5kg and 2.5kg) and normal birthweight (above 2.5kg) (WHO, 2019).

- **History of asthma in the family**

Epidemiological studies have shown evidence that hereditary factors are important contributors of asthma (Lim et al., 2010; Civelek et al., 2010). In MCS, the main respondent (who has been restricted to be the birth mother in the statistical analysis of this thesis) and partner (who was the child's father in the majority of cases[17]) were asked "*Have you ever been told by a doctor or nurse that you had asthma*?". Responses from this question were used to categorise children into two categories "Family history of asthma" or "No Family history of asthma", with the latter being the reference category. Family history of asthma was included as an a priori variable as it is intended to provide a proxy for asthma heritability and is unchangeable.

[17] Only cases where the father was the natural father were included

- **Number of siblings**

Number of siblings was included as an a priori explanatory variable as it is an unchangeable factor at a given age (fixed at the age at which it is measured, i.e. 9 months). It could also be considered an explanatory variable owing to the link between risk of asthma-related outcomes and exposure to siblings or other children. One explanation for this is the hygiene hypothesis which claims that reduced exposure to early life infection and microbial exposure through more thorough hygiene levels and fewer siblings in the home (both of which influence transmission pathways for infection) impedes natural immune system development (Azad et al., 2013). This in turn may lead to a predisposition to as asthma and wheezing, but there is mixed evidence in support of the hygiene hypothesis. In the MCS, main respondents are asked how many siblings the cohort child had at 9 months. In the statistical analysis, sibling categories included are: no siblings, one, two, and three or more siblings, with no siblings being the reference category.

2.10.2. Other potential explanatory variables

These are factors that have been identified in the literature that may explain, to some extent, the variation in asthma-related outcomes amongst children, as described in Section 1.5, Chapter 1. Certain explanatory variables may act as a channel by which low income translates into poor asthma related-outcomes (Propper et al., 2006; Violato et al., 2009; Kuehnle, 2014). It is important to note however, that there is no clear distinction in the literature between the factors that are included as a priori variables and those that are included as explanatory variables (Propper et al., 2006).

2.10.2.1. Other measures of socioeconomic status

- **Mother's education**

Parental education is often included as a measure of socioeconomic status in epidemiological and economic studies of child health, either with or in place of income (Case et al., 2002; Propper et al., 2007; Taylor-Robinson et al., 2015). Additionally, mother's education specifically, has been found to be significantly associated with asthma-related outcomes (Violato et al., 2009; Taylor-Robinson et al., 2015). Mother's education may be potentially one of the most important social determinants of child health (Lewis et al., 2017). Mothers with lower education often face systematic barriers, such as to the employment market or jobs with poor working conditions, which may result in less time or resource to put into caring for their children, reflecting in poor child health outcomes. It has also been argued that to omit mother's education with the knowledge that it could be highly correlated with income, would result in an overestimation of the effect of income – an omitted variable bias (Propper et al., 2007).

In MCS 1, mother's highest level of education is derived from questions about academic and vocational qualifications. Education is then recorded as one of the NVQ levels 1-5, overseas qualification or no qualification. NVQ categorisation is constructed to include equivalent academic qualifications: NVQ 1 – General Certificate of Secondary Education (GCSE) grade D-G or equivalent; NVQ 2 – GCSE A*-C or equivalent; NVQ 3 – A levels or equivalent; NVQ 4 – Higher National Diploma, bachelor degree or equivalent; NVQ 5 – Post graduate degree level. NVQ 1 is the reference category.

- **Housing tenure**

There is evidence of housing tenure having an influence on discrepancies in child health outcomes, where children living in social or council housing are more likely to be admitted to hospital, compared to children living in privately rented or owned homes (Krieger, 2010; Nasim, 2015). The reasoning is that housing tenure may capture housing quality, to some extent. For example, council housing might be linked to areas with a lot of pollution or area deprivation

(Krieger, 2010; Kuehnle 2014). Housing tenure may also capture socioeconomic status to some extent, whereby families in higher socioeconomic groups are more likely to live in owned homes than lower socioeconomic groups. Housing tenure is therefore an important determinant of child health and potentially asthma-related outcomes, where its omission could lead to potential overestimation of the income effect (Kuehnle, 2014). Housing tenure is recorded in MCS 1 at the age 9 months survey and then the question repeated at each subsequent survey only if housing status had changed. The question asked was "*Do you [^or your husband/wife] own or rent your home or have some other arrangement?*". The housing variable used in the analysis is derived from this question and comprises three categories, namely "Mortgaged or owned", "Rented from the council", "Rented privately or from housing association". The category "mortgage or owned" is the reference category.

- **Mother's age at birth of cohort child**

Women with higher education tend to defer motherhood until later, compared to women with lower levels of education (Propper et al., 2006; van Roode et al., 2017). There is also evidence that older mothers may have a larger wealth of health knowledge including health literacy, which they then use in prevention and/or management of disease in their children (Laerum et al., 2007). In MCS 1, mothers are asked their age at their last birthday. For the statistical analysis, mothers are then placed into one of the following age brackets in years: <20, 20 to 24, 25 to 29, 30 to 34, 35 to 39 and age 40 and above, where <20 is the reference category.

2.10.2.2. Mother and child health-related behaviour

- **Alcohol consumption during pregnancy**

The literature shows that alcohol consumption during pregnancy is associated with poorer outcomes in early childhood, such as preterm birth and low birthweight; however, there has not been conclusive evidence to show that alcohol consumption is associated specifically with

asthma-related outcomes in childhood. Nonetheless, this association may exist through low birthweight which itself strongly associated with an increased risk of asthma (Magnus et al., 2011; Miyake et al., 2008). In MCS 1, mothers were asked to select the frequency of their alcohol consumption during pregnancy. Categories included consumption 'every day', 5-6 days per week, 3-4 days per week, 1-2 days per week, 1-2 times per month, less than once per month and never. Mothers were then asked how many units[18] of alcohol they consumed in general, based on their responses. For the purposes of the statistical analysis, the UK National Alcohol Strategy for heavy drinking categories are applied (Kelly et al., 2012). Categories included are: 'never drank alcohol', 'light consumption' (1-2 units a week), 'moderate consumption' (3-6 units a week) or 'heavy consumption' (7 or more units a week). 'never drank alcohol' is the reference category.

- **Maternal smoking in pregnancy**

Epidemiological literature has shown that smoking during pregnancy is one of the most modifiable risk factors of childhood asthma. This is due to the direct damage on foetal lung development, although it has also been found to increase the risk of wheezing in infants (Neuman et al., 2012; Zacharasiewicz, 2016). As an important risk factor for asthma-related outcomes, smoking during pregnancy is included in the analysis. In MCS 1, mothers were asked a series of questions about their smoking habits:

"*How many cigarettes a day were you smoking just before you became pregnant?*", "*And did you change the amount you smoked during your pregnancy?*", "*In what month of pregnancy did you make this change?*" and "*How many cigarettes a day did you usually smoke after you made this change?*".

[18] One unit of alcohol corresponded to: half a pint of beer, a glass of wine, or a single measure of spirit or liqueur

The variable for smoking during pregnancy is then categorised as follows: 'never smoked', 'quit smoking' [at the beginning of the pregnancy], 'kept smoking' [during pregnancy] with 'never smoked' as the reference category.

- **Mode of delivery**

Evidence from the literature suggests that there are long term health effects, such as an increased risk of asthma, that are associated with delivery by caesarean section compared to normal vaginal delivery (Keag et al., 2018). In MCS 1, mothers were asked "*What type of delivery did you have?*". They could then select one response from: 'a normal delivery', 'assisted with forceps', 'assisted vacuum extraction', 'assisted breach', 'a planned caesarean', 'emergency caesarean', or 'other'. For this thesis, the above categories are more narrowly classified into: 'normal delivery', 'assisted delivery' (this included assisted with forceps, vacuum extraction or other assisted), 'planned c-section' and 'emergency c-section'. 'Normal delivery' is the reference category.

- **Mother's BMI at birth of child**

Maternal obesity and high weight gain during pregnancy, termed 'gestational weight gain', have both been found to be associated with the development of asthma in childhood (Forno and Celedon, 2009; Polinski et al., 2017). Likewise, very low weight gain during pregnancy has also been associated with asthma in childhood (Forno and Celedon, 2009; Polinski et al., 2017). In MCS 2, mothers were asked about their weight and height at the birth of their child. For the purpose of my thesis, I classified mother's body mass index (BMI[19]) into the following categories: 'underweight' (15-19.9 kg/m^2), 'normal weight' (20-24.9 kg/m^2), 'overweight and

[19] Body Mass Index is calculated as: weight (kg)/square of height (m^2)

obese' (25-29.9 and >30 kg/m^2), according to the WHO classification (Nuttall, 2015). 'Normal weight' is the reference category.

- **Mother's general health**

Mother's health could have a direct effect on child health whereby a mother is less able to care for her child due to her own illness, in turn affecting the child's health (Case et al., 2002). There may also be a genetic link or an inherited susceptibility to disease, for example, the mother and child may both have asthma. Finally, there may be unobserved factors generating a spurious effect between maternal health and child health (Case et al., 2002; Kuehnle 2014). Overall, the literature shows that mothers who are in poor general health or have a chronic health condition are more likely to have a child who is in poor health (Currie et al., 2007). In light of this evidence, mother's health is included as an explanatory variable. In MCS 2-5 mothers were asked by the interviewer:

"*I would now like to ask about your health. How would you describe your health generally? Would you say it is excellent, good, fair or poor?*".

For the purposes of the analysis, the four response options are grouped into two general health categories due to small numbers of women reporting excellent health and poor health, compared to good and fair health. Categories are "Good or excellent health" and "Fair or poor health" with the former being the reference category.

- **Mother's mental health**

As with general health, poor maternal mental health may also have an adverse effect on child health by lowering the quality of care for the child through similar mechanisms as for general health (Meyer, 2002). Mothers who are suffering anxiety or depression, for example, may be less able to manage their children's health conditions. It has also been shown that treating mothers for depression has a significant improvement in paediatric asthma (Ghaempanah et al.,

2013). In light of the growing diagnosis of mental health problems (especially amongst mothers with young children), mother's mental health is an important explanatory variable in its own regard, in addition to general health.

In MCS 2-5 mothers were asked six questions adapted from the Kessler Screening Test for Psychological distress (referred to as the K6), about how often they felt sad, nervous, restless or fidgety, hopeless, that everything is an effort and worthless in the last 30 days (Kessler et al., 2002; Mitchell and Beals., 2011). Responses ranged from 0 (none of the time) to 4 (all of the time). K6 scores were then summed across the six items resulting in a total score range of 0 to 24. I then categorised K6 scores using standard cut-offs, into 'no or low distress' (score<=3), 'medium distress' (score 4-12) or 'high distress' (score >12). 'No or low distress' is the reference category (Kessler et al., 2002).

- **Breastfeeding**

There have been numerous epidemiological studies investigating the effect of breastfeeding on asthma-related child health outcomes (Dogaru et al., 2014; Lodge et al., 2014; Quigley et al., 2018). From systematic reviews and meta-analyses, evidence has been found to support that breastfeeding is protective for asthma. In general, the protective effect of breastfeeding for asthma is acknowledged but the extent to which it is protective, or the generalisability of the effect is difficult to assess due to differences in measurement of child asthma-related health outcomes, and a large spread of breastfeeding patterns (Quigley and McGuire, 2014). In MCS 1, mothers were asked of their child: "*Did you ever try to breast feed him/her?" and "How old was the child when he/she last had breast milk?*"

From these questions breastfeeding duration was calculated and categorised into 'never breastfed', [breastfed for] 'less than 2 months', '2 to 4 months', '4 to 6 months' and 'more than 6 months' with 'never breastfed' as the reference category.

2.10.2.3. Household and environmental factors

- **Damp or condensation in the home**

It has been observed that asthma and wheezing in children are triggered when they live in homes that are damp, one of many established asthma triggers (Andriessen et al., 1998; Fisk et al., 2007; Thacher et al., 2017; Asthma UK, 2019). Damp in the home can also attract mould spores, which are associated with exacerbated asthma and wheezing (Asthma UK, 2019). In the MCS, main respondents are asked if there was damp or condensation in the home when the child was 9 months, and this is operationalised as a dichotomous variable in my statistical analysis, with 'no damp' as the reference category.

- **Exposure to fumes from household heating**

Fumes from open fires, wood-burning stoves or other fuel-based heating can aggravate asthma symptoms in some people, including young children. This is because burning wood, coal or other fuel can release fine particles and fumes, which when inhaled can make the airways inflamed (Belanger and Triche., 2008; Asthma UK, 2019). In the MCS 1, main respondents are asked about the type of heating they used in the home. I categorised responses by heating method, depending on whether they were likely to produce fine particles or fumes (coal, gas, wood fires and paraffin heaters produce fumes and central heating and electric fires and heaters do not), creating a dichotomous variable 'no exposure to fumes from heating' or 'exposure to fumes from heating', with the latter as the reference category.

- **Exposure to cigarette smoke**

Many epidemiological studies have highlighted the strong association between second hand smoke and asthma and wheezing in children (Tinuoye et al., 2013). Tobacco smoke irritates the airways, subsequently triggering asthma, and more strongly in children (Asthma UK, 2019). In

MCS 1, respondents are asked whether anyone in the household smoked in the same room as the child in the first 9 months; from this I generated a dichotomous variable called 'exposure to cigarette smoke at 9 months', with 'no exposure' as the reference category.

- **Rural-urban morphology**

There has been evidence to suggest that urbanisation is a risk factor for asthma and wheezing in children, although not conclusive in the UK, with previous findings showing that children in urban areas have worse asthma-related outcomes compared to their rural counterparts, due to better air quality (Asher, 2011; Malik et al., 2012; Estrada and Ownby, 2017). Rural areas tend to be less densely populated, with less vehicle traffic compared to urban areas which tend to have the prototype environment to trigger asthma. In the MCS, cohort members are assigned a rural-urban morphology code[20]. For my analyses, I created three categories: 'urban', 'rural' or in a 'rural-urban mix', with 'urban' as the reference category.

2.11. Statistical analysis

This section provides the theoretical model that provides the basis for the model estimations throughout this thesis. It also gives an overview of some of the statistical methods used. More specific details of methods and variables used are included within their respective results chapters. All analyses in this thesis were undertaken in Stata14.

2.11.1. Theoretical model

[20] Codes differed between the UK countries with England, Wales and Northern Ireland having three similar categories but Scotland having six. For Scotland "large" and "other" urban areas were classified under urban for my analyses, accessible and small towns were classified as mixed, and accessible and remote rural was classified as rural.

The theory applied throughout this thesis is based on Grossman's human capital model of the demand for health (Grossman, 1972; Grossman, 2000). This model is so called as it is largely based on the human capital theory which states that:

"*increases in an individual's stock of knowledge or health capital raises his productivity in the market sector of the economy where he produces money earnings and in the non-market sector where he produces commodities that enter his utility function*" (Becker, 1964)

Grossman (1972) was the first to construct a model of the demand for health capital itself. The Grossman theory of health accumulation assumes that individuals inherit an initial stock of health, and that health can be increased by investments which can be seen as factors which are used to produce health. Adapted to the case of children, we can refer to a 'child-health production function', as first applied by Case et al (2002). In this version of the model, child-health is determined by 1) parental income 2) a set of parental factors 3) child characteristics 4) initial child health endowment. The general child-health production function can be expressed as follows:

$$H_{it}^{c} = \alpha + \beta lnY_{it} + \gamma X_{it}^{p} + \theta X_{it}^{C} + h_{i0}^{c} + \varepsilon_{it} \quad (2.1)$$

where:

H_{it}^{c} is child *i*'s health at time *t*, Y_{it} is family income; X_{it}^{p} is a vector of parental inputs other than income, and X_{it}^{C} is a vector of child characteristics; h_{i0}^{c} is the initial stock of child health and ε_{it} the residual. In addition, family income itself is determined by the following equation:

$$Y_{it} = \emptyset + \varphi Z_{it} + \delta h_{it}^{p} + \mu_{it} \quad (2.2)$$

where

Z_{it} represents a set of time varying and time invariant parental characteristics and $h_{it}^{p}$is parental health and μ_{it} is the residual.

Equation (2.1) is adapted as necessary within the thesis to represent a child's 'asthma-related outcomes' version of the health production function. Equation (2.2) is encompassed within equation (2.1) by including variables that account for parental characteristics. Adaptations of the child-health production can be found within each relevant chapter.

2.11.2. Estimation methods

2.11.2.1. Logistic regression

In the cross-sectional analysis (Chapter 3), binary asthma-related outcomes are estimated. Because of the binary nature of the asthma-related outcomes (taking on values of 0 or 1), the standard OLS estimation is not suitable. Instead, a logistic regression model is fitted, and the parameters for this model are estimated using maximum likelihood estimation (MLE) (Rodriguez, 2007). The estimates can either be displayed as ORs or logistic coefficients (log ORs) of which the marginal effect may be estimated for more intuitive interpretation (details of this in Chapter 3).

2.11.2.2. Multinomial regression

In Chapter 4, the wheezing outcome is measured in categories based on onset and duration, taking on one of four possible phenotypes which are defined in Chapter 4. The logistic regression model is extended to include the case where the dependent variable takes on more than two categories in a multinomial regression. Like the general logistic regression, it also uses MLE to evaluate the probability of categorical membership. Relative Risk Ratios (RRR) and logistic coefficients with marginal effects can both be estimated. Both logistic and multinomial

logistic regression in Chapter 3 and 4 used the survey commands in Stata to incorporate the clustering and stratification of the MCS survey design.

2.11.2.3. Panel data methods

The longitudinal analysis in Chapter 5 included panel data methods - fixed effects (FE) and random effects (RE) models were estimated. In the former, time-invariant characteristics are removed and unobserved variation does not change over time, therefore enabling the net effect of family income and on asthma-related outcomes to be assessed. Because the survey properties cannot be incorporated into the analysis, due to limitation of the software, alternative longitudinal techniques were implemented. Specific details of the methodology together with the estimation strategy are explained in more detail within Chapter 5.

2.11.2.4. Decomposition of the Concentration Index

The final results chapter of this thesis, Chapter 6, focusses on inequality and estimates indices of income-related health inequality in asthma-related outcomes. These estimated indices of inequality are then decomposed into contributing factors, giving a comprehensive description of the contribution of determinant factors towards overall inequality. Specific details of the methodology are explained in Chapter 6.

3.Cross-sectional analysis

3.1. Introduction

The relationship between income and child health has been investigated in children from a variety of age groups and countries including the UK, as was seen in the literature review in Chapter 1. It is well established, across different child population groups, that children from families with a low socioeconomic status tend to have poorer health than children from higher socioeconomic groups (Case et al., 2002; Currie and Stabile, 2003; Currie et al., 2007; Condliffe and Link, 2008; Khanam et al., 2009; Reinhold and Jürges, 2012). However, whether this relationship persists as children age still remains inconclusive, particularly in the UK.

Whether the association between socioeconomic status and child health exists in specific health areas such as child asthma-related outcomes has been investigated in a few studies to date (Violato et al., 2009; Hafkamp de Groen et al., 2013; Taylor- Robinson et al., 2015; Galobardes et al., 2015). The focus on asthma-related outcomes in this thesis is driven by the high prevalence of conditions such as asthma and wheezing in children in the UK where 1 in 11 children suffer from asthma, representing one of the highest rates in Europe (Asthma UK, 2018). The focus on the association between income and asthma-related outcomes in childhood is further motivated by the UK having one of the highest levels of children who live in low income families in Western Europe. In 2017, there were 4.1 million children in the UK (30% of children) living in defined poverty compared to an average of 5% across western Europe with child poverty in the UK having worsened in the last decade (Brewer et al, 2011; CPAG, 2016).

The objective of this chapter was to examine the association between family income and asthma-related outcomes in children at ages 3, 5, 7 and 11[21] years using data from the MCS (Objective 1). Where low income is significantly associated with asthma-related outcomes, this can be referred to as the 'child health- income gradient'. In addition to income, the association between other factors and asthma-related outcomes was also explored given the rich dataset and association identified in the literature. These other factors include: a priori variables, other socioeconomic factors, mother and child health-related variables and environmental and household characteristics. The second objective was to explore how the child health-income gradient might change as children grow older (Objective 2). I would expect that the gradient strengthens with age as children might have a higher rate of health shocks as they grow older and that effects of poor health in early childhood are long lasting and become more evident in later childhood.

3.2. Methods

3.2.1. Analytical framework & model specification

Child asthma-related outcomes were estimated using a production function informed by the health capital model developed by Grossman (1972a, 1972b and 2000) (Chapter 2, Section 2.11.1). Equation (3.0) was used to estimate the raw association between income and asthma-related outcomes. Equation (3.1) represents the equation used to model asthma-related outcomes in children, explanatory variables were then subsequently added to equation (3.0). Child *i's* asthma-related outcome at time *t* can be expressed as:

[21] Asthma-related outcomes were not analysed at age 9 months in the main analysis as these outcomes were not consistent with the age 3, 5, 7 and 11 asthma and wheezing outcomes. However, I performed an exploratory analysis using data from age 9 months. Results are in Appendix B-1.

$$H_{it}^{c} = \alpha + \beta lnY_{it} + \varepsilon_{it} \quad (3.0)$$

$$H_{it}^{c} = \alpha + \beta lnY_{it} + \gamma X_{it}^{p} + \theta X_{it}^{C} + \varepsilon_{it} \quad (3.1)$$

Where H_{it}^{c} is child *i*'s asthma-related outcome at time *t*, Y_{it} is family income for child *i* at time *t*, X_{it}^{p} is a vector of parental characteristics, and X_{it}^{C} is a vector of child characteristics. γ and θ represent the vectors of parameters to be estimated. ε_{it} is the random error.

3.2.2. Description of variables

3.2.2.1. Asthma-related outcomes

Three asthma-related outcomes health outcomes were used in the cross-sectional analysis: ever asthma, wheezing in the last 12 months and asthma medication use. These were derived as binary variables (1=Yes; 0= No) from the responses to three specific MCS questions that are summarised in Table 3.1.

Table 3.1: MCS questions relating to asthma and wheezing

Outcome	Question	Age 3	Age 5	Age 7	Age 11
Ever asthma (Yes/No)	Has the child ever had asthma?	✓	✓	✓	✓
Wheezing (Yes/No)	Has the child ever had whistling or wheezing in the last 12 months?	✓	✓	✓	✓
Asthma medication use (Yes/No)	Does the child take any regular medication? Follow up: What medication?	×	✓	✓	✓

3.2.2.2. Income

The main exposure variable in the cross-sectional analysis was family income, which was operationalised as the natural logarithm of permanent income. To recap, permanent income

was calculated as the average household income up to and including income at the age at which the asthma-related outcome is measured (Chapter 2, Section 2.9.1).

3.2.2.3. Other explanatory variables

To account for the parent and child characteristics in the analytical framework, four groups of explanatory variables were also included in the analysis. These variables were informed by literature review prior to the empirical analysis. These were grouped as: a priori variables, other measures of socioeconomic status, mother and child health-related variables and environmental and household factors (Chapter, Section 2.10.).

3.2.3. Estimation strategy

A five-stage stepwise regression analysis that included five model specifications was implemented for each asthma-related outcome, i.e. ever asthma, wheezing in the last 12 months and asthma medication use. The first stage estimated the unadjusted regression coefficient between income and asthma-related outcomes. At each of the five subsequent stages, the set of variables associated with that step was added to the model. Table 3.2 outlines how explanatory variables were included in the five-stage stepwise regression analysis.

Table 3.2: Model specification for cross-sectional analysis

Model specification	Description of variables included in models
Specification 1	Unadjusted association between income and asthma-related variables
Specification 2	Specification 1+ a priori variables: ethnicity, sex, child age birthweight family history of asthma Number of siblings at 9 months
Specification 3	Specification 2 + other socioeconomic variables:

Model specification	Description of variables included in models
	mother's highest level of education at 9 months+ mother's age at time of birth + housing tenure (t-1)[22]
Specification 4	Specification 3+ mother and child health-related variables: mother smoking during pregnancy + mother's alcohol consumption during pregnancy + mother's BMI at birth of child + delivery mode + breast feeding duration + mother's general health (t-1) + mother's mental health (t-1)
Specification 5	Specification 4 + household and environmental factors: damp or condensation in the home at 9 months + exposure to cigarette smoke at 9 months + exposure to fumes from heating at 9 months + rural-urban morphology at 9 months

A priori confounders were included at each step because their association with asthma-related outcomes, if any, should be considered so as not to overestimate the magnitude of the association between income and the asthma-related outcomes by omitted variable bias. Mother's education was also included in model specification (3-5) as it is an important determinant, widely used in epidemiological and economic literature as a measure of socioeconomic status. The other explanatory variables, introduced in Specification 3, 4 and 5 (Table 3.2) only remained in the model if they were significantly associated with the outcome variable (Wald test p-value<0.1) after adjusting for all other variables in that model specification. All models were estimated using logistic regression (Chapter 2, Section 2.11.2.1). Results were expressed in terms of marginal effects of estimated logistic coefficients which allow a quantitative interpretation and are widely used in health economics literature. Results were also expressed as odds ratios (ORs) which are useful for comparison with published epidemiological literature.

Marginal effects measure the change in the probability of a child having a specific asthma-related outcome that is produced by a change in income (Cameron and Trivedi, 2010). Marginal effects for categorical explanatory variables measure discrete change and express how predicted probabilities of asthma-related outcomes change as the explanatory variable changes relative to the reference category. As income was operationalised as a natural logarithm within a logistic

[22] t-1 indicates the MCS cohort directly preceding MCS t

model, the marginal effect of income could be interpreted as a semi-elasticity – the change in probability of reporting a specific health outcome associated with a 1% increase in income. There are several different partial effects that can be estimated but in this analysis, average marginal effects[23] (AMEs) have been selected as they consider the entire sample population used in the analysis (Cameron and Trivedi, 2010). The margins 'dydx' command was used to generate AMEs.

ORs were derived from logistic models and are interpreted as the estimated change in odds of the asthma-related outcome associated with a 1% increase in income. If the OR is equal to one, then the change in income does not affect the odds of the outcome occurring. If the OR is less than one, then a 1% increase in income is associated with a decrease in odds of the outcome, and if the OR is more than one, then a 1% increase in income is associated with an increase in the odds of the outcome occurring (Szumilas, 2010).

All analyses were repeated for ages 3, 5, 7 and 11 for the following outcomes: ever asthma, wheezing in the last 12 months and asthma medication use (ages 5, 7 and 11). The clustered, stratified study design and sample weights were taken into account in the analyses using 'svy' commands.

3.2.4. Sensitivity analysis

As a sensitivity analysis, lagged income was used as an alternative to permanent income. An identical empirical strategy was applied and results can therefore be compared. Lagged income was calculated as the natural logarithm of annual family income in the survey year preceding the year at which the asthma-related outcome was measured, inflated to 2011 prices (Chapter 2, Section 2.9.1). As with the main analysis, the sensitivity analysis were repeated for ages 3, 5, 7

[23] AME measures the average change in probability when x increases by 1 unit.

and 11 for ever asthma, wheezing in the last 12 months and asthma medication use (ages 5, 7 and 11).

3.3. Results

3.3.1. Overview of the data

The final sample included 10,260 singleton children present throughout MCS 1-5. Table 3.3 shows mean annual family income for each survey age. Figures reported are in pound sterling (£) values and are in 2011 prices. These figures show how permanent income varies between income quintiles with highest income quintiles having up to six times more income than the lowest quintile. At age 11 mean annual income may be higher due to enhanced collection of income data in the survey (Platt, 2014).

Table 3.3: Mean annual family income (£) for all ages by income quintile

	9 months	Age 3	Age 5	Age 7	Age 11
Lowest quintile	7,103	6,665	7,409	7,515	12,919
Second quintile	13,192	13,436	13,350	13,482	19,790
Third quintile	19,848	20,471	19,784	19,560	26,910
Forth quintile	28,216	28,957	27,632	27,056	34,135
Highest quintile	46,913	47,547	44,597	42,837	44,733
Overall	23,159	23,635	22,985	22,527	28,565

Figure 3.1 gives a graphical representation of mean income per income quintile in 2011 prices, a fairly constant distribution of income across quintiles at all ages can be seen. Figure 3.2 shows the distribution of family income according to ever asthma in the MCS in children at age 3, 5, 7 and 11. At all ages, the median and upper quartile of family income are higher for children in the never asthma group compared to those in the ever asthma group. This was the same in the case of wheezing in the last 12 months (Figure 3.3) and asthma medication use (Figure 3.4).



Figure 3.1 Income quintile by age

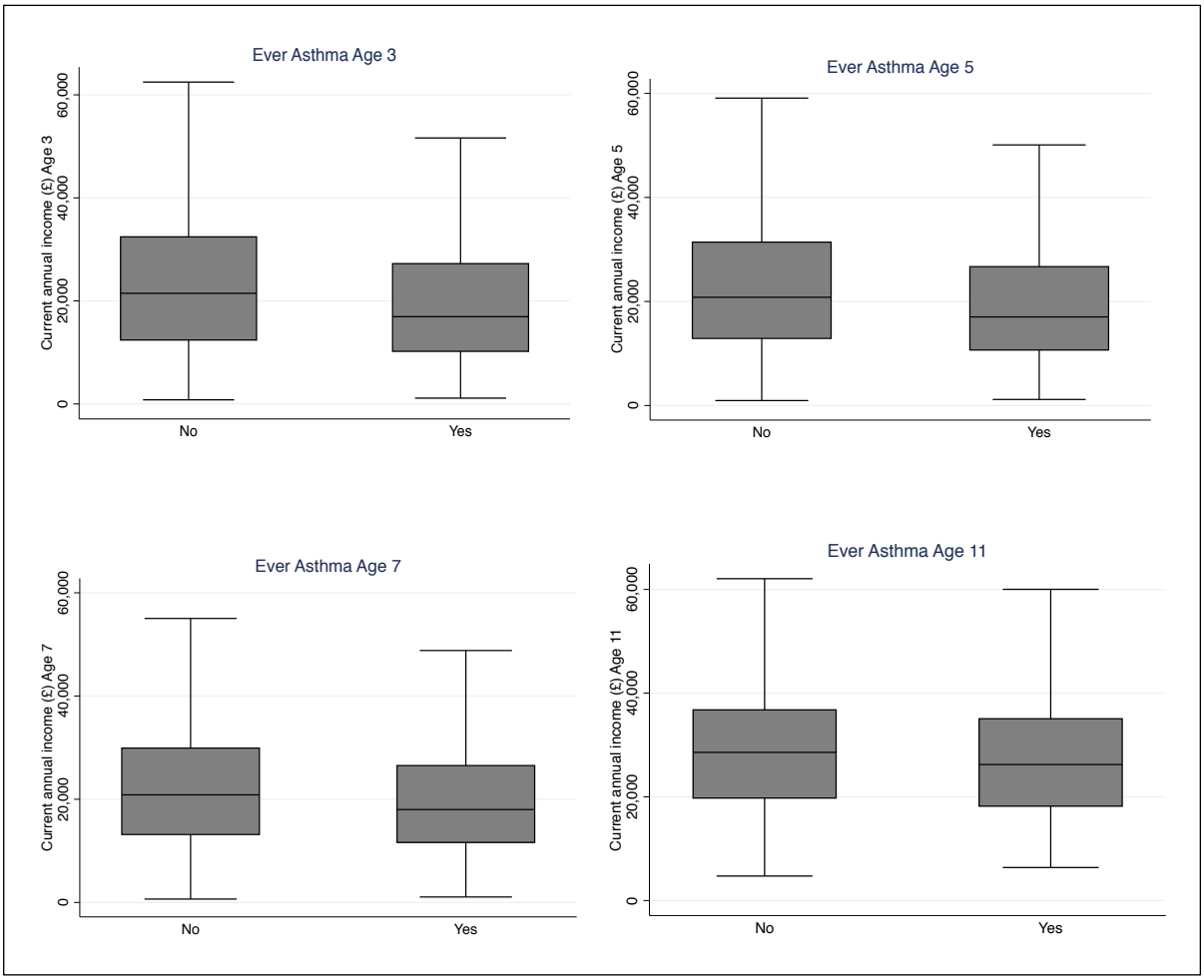


Figure 3.2: Median and interquartile range of family income: Ever asthma

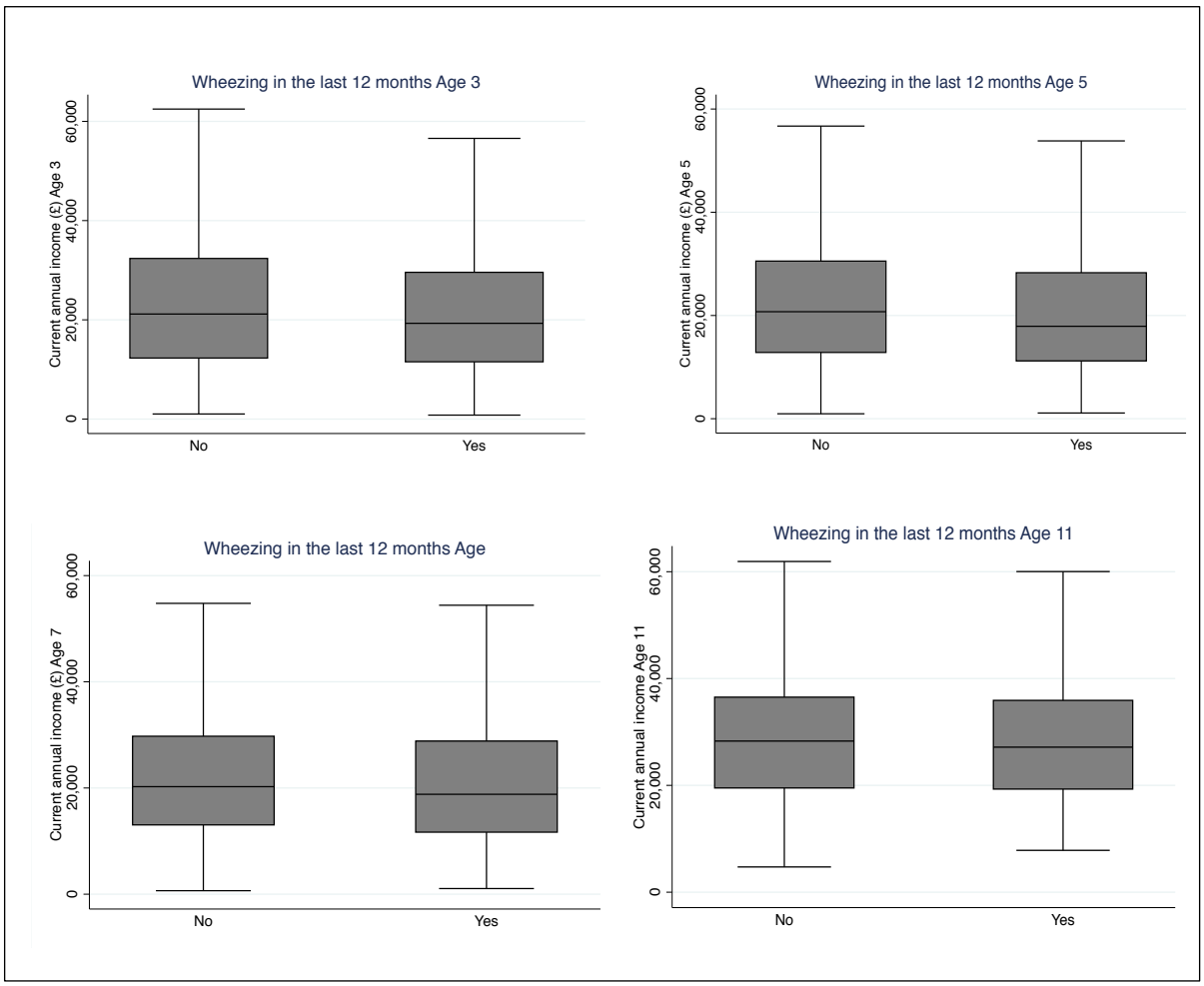
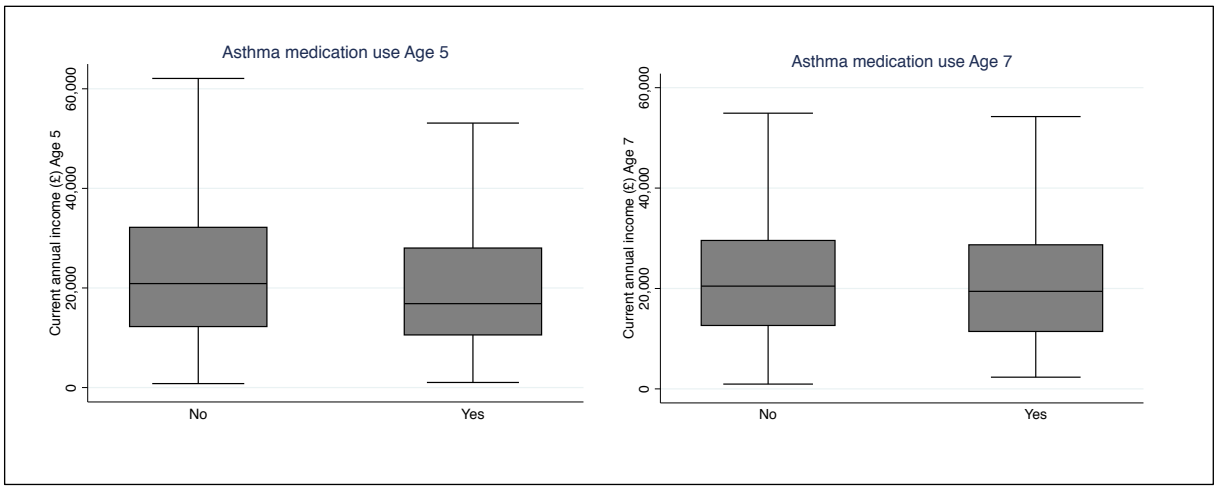


Figure 3.3: Median and interquartile range of family income: Wheezing in the last 12 months



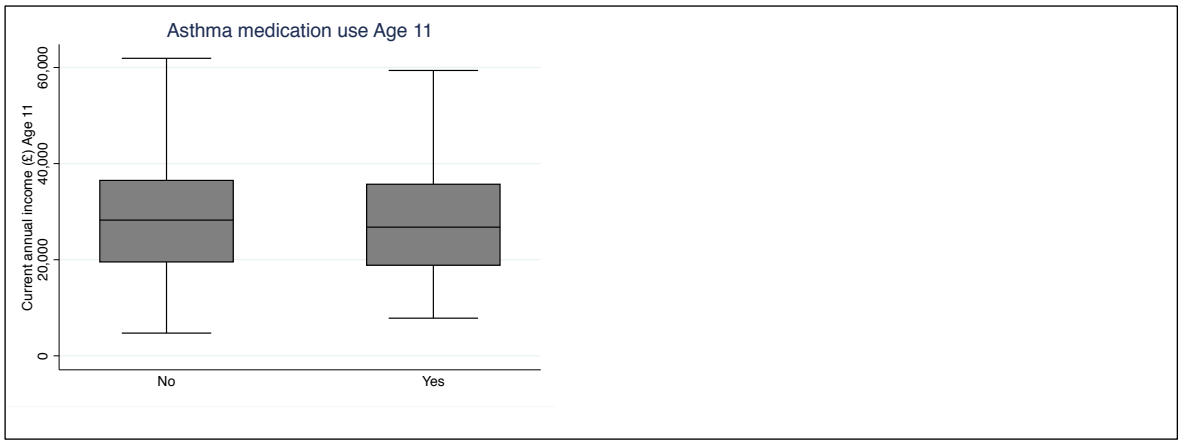


Figure 3.4: Median and interquartile range: Asthma medication use

Table 3.4 shows the distribution of all a priori and explanatory variables across income quintiles used throughout in this thesis. The bivariate association between each potential confounder and income was tested through a Chi-square test and the resulting p-values are reported.

In terms of child demographics, 49.8% of the sample was male. Sex was not significantly associated with income and the proportion of males and females in each income quintile, was similar. Almost 89% of the sample children were white, with Pakistani being the next largest ethnic group (3.3%). Ethnicity was significantly associated with income (p-value:0.01). Only 15% of white children were in the lowest income group whereas 22.6% were in the highest income group. In contrast, 66.9% of Pakistani, 75.9% of Bangladeshi, 41.4% of black Caribbean and 45.1% of black African children were in the lowest income groups with a small proportion of these groups belonging to the high-income group (2.8% for Pakistani and 1.7% for Bangladeshi children).

Birthweight was strongly associated with income with 25.4% of low birthweight children falling in the lowest income group, whereas only 13.9% were in the highest income group. Preterm birth follows a similar pattern although the association was not as strong. Overall, 26.3% of children had a family history of asthma and were less likely to be in the lowest income quintile (16.3%) than the highest (20.9%). Number of siblings was also negatively associated with

income level – about 50% of families with one or two children (i.e. 0-1 siblings) fell into the top income quintiles, whereas families with three children (2 or more siblings) fell into the bottom three income quintiles (p-value<0.001). Over 45% of children with three or more siblings fell into the lowest income quintile (p-value<0.001).

There was a clear pattern in terms of mother's age at time of birth and income level, with younger mothers more likely to fall into the lower income quintiles compared to older mothers (p-value <0.001). For example, mothers in age categories age '14-19' and 'age 20-24' were more likely to be in the lowest income quintile (56.5% and 35.3% respectively), whereas mothers aged 30 and above, were more likely to be in the highest income quintile (31.8%, 36.0% and 34.9% respectively). Mother's education was positively associated with income level (p-value< 0.001). 35% of mothers with no education fell into the lowest income quintile compared with 1.1% of mothers with a postgraduate degree (NVQ level 5).

Analysing mother and child health-related behaviour, it can be seen that light and moderate alcohol consumption during pregnancy were more frequent in mothers from the top two income quintiles (p-value<0.001). In contrast, mothers who reported heavy drinking during their pregnancy were mostly from the lowest income group. Similarly, with respect to smoking during pregnancy, children whose mothers never smoked before their pregnancy were concentrated in higher income groups in comparison to those whose mothers continued smoking during their pregnancy who were mostly in the bottom two income quintiles. Children born to mothers who were obese or underweight (BMI >29.9 kg/m^2 or <18.5 kg/m^2 respectively), were mostly from low-income families whereas children born to mothers with a normal BMI tended to be in the top three income quintiles (p-value<0.001). Children born by assisted delivery, planned and emergency c-section were least concentrated in the bottom two income quintiles whereas children born by normal delivery were generally evenly distributed amongst the income quintiles (p-value<0.001).

Breastfeeding was strongly associated with family income (p-value<0.001). Breastfeeding for two or more months was more common in mothers from higher income families, whereas children who were never breastfed are highly concentrated in the bottom two income quintiles (30% for the lowest income quintile and 25.9% for the second lowest quintile). In terms of maternal health, mothers with good or excellent general health and good mental health (low levels of distress) were concentrated in higher income quintiles whilst mothers with poor general and poor mental health belonged to the low income quintiles (45% of mothers with high levels of distress were also in the lowest income quintile; p-value <0.001).

Environmental and household factors indicated that children exposed to cigarette smoke and who lived in homes that had damp were generally from low-income families (p-value<0.001). Children who lived in rural and mixed rural-urban environments at 9 months tended to belong to the top three income quintiles (p-value<0.001).

Table 3.4: Summary of explanatory variables from the MCS distributed by income quintile

	Total	Total %	Quintile					P-value
			Lowest	2nd	3rd	4th	Highest	
			%	%	%	%	%	
Child Characteristics:								
Child sex								
Male	4884	49.8	17.9	18.7	20.6	21.3	21.5	0.499
Female	4922	50.2	18.8	19.8	19.9	20.4	21.1	
Ethnicity								
White	8698	88.9	15.0	19.1	21.1	22.2	22.6	<0.001
Mixed	80	0.8	34.0	28.1	12.4	14.1	11.4	
Indian	186	1.9	11.1	28.8	24.9	13.2	22.0	
Pakistani	325	3.3	66.9	19.2	8.0	3.2	2.8	
Bangladeshi	105	1.1	75.9	14.9	3.1	4.3	1.7	
Black Caribbean	98	1.0	41.4	15.5	18.9	10.9	13.3	
Black African	136	1.4	45.1	14.8	16.6	9.2	14.3	
Other	158	1.6	29.0	22.2	11.8	20.7	16.4	
Birth weight, kg								
Normal (>2.5)	9188	93.7	17.9	18.9	20.3	21.1	21.8	<0.001
Low (1.5kg < & ≤ 2.5)	538	5.5	25.4	25.3	18.4	17.0	13.9	
Very low (≤1.5)	80	0.8	21.5	23.3	21.1	20.8	13.4	
Gestation, weeks								
Term (>37)	8887	90.6	18.0	19.1	20.4	20.7	21.7	0.048
Preterm (32 < & ≤ 37)	827	8.4	21.6	19.6	18.8	22.1	17.9	
Very preterm (≤ 32)	92	1.0	20.0	29.5	19.3	19.9	11.5	
History of asthma in family								
No	7204	73.7	19.1	18.3	20.1	21.0	21.5	0.002
Yes	2602	26.3	16.3	22.1	20.5	20.3	20.9	
Number of siblings at 9 months								
None	4006	41.7	15.1	17.4	18.9	23.2	25.4	<0.001
One	3667	37.0	14.8	19.0	20.8	22.7	22.7	
Two	1457	14.5	23.6	24.3	22.1	16.0	13.9	
Three or more	677	6.8	45.6	20.9	21.1	7.1	5.4	
Maternal characteristics								
Mother age at birth of child, years								
14-19	677	6.7	56.5	31.7	9.3	2.3	0.2	<0.001
20-24	1578	15.8	35.3	32.9	19.4	9.2	3.2	
25-29	2859	28.9	16.2	19.0	24.4	23.8	16.6	
30-34	2999	30.8	8.6	12.9	19.8	26.9	31.8	
35-39	1452	15.3	8.1	12.2	19.0	24.7	36.0	
≥40	241	2.5	7.6	20.6	21.3	15.6	34.9	
Education level								
NVQ level 1 (GCSE D-G)	823	8.3	35.7	31.2	19.6	9.1	4.3	<0.001
NVQ level 2 (GCSE A*-C)	2947	29.6	17.5	25.3	26.4	20.9	9.9	
NVQ level 3 (AS and A level)	1450	14.5	8.6	22.2	27.3	25.3	16.6	
NVQ level 4 (Bachelor degree)	2873	30.1	2.2	7.0	16.8	30.3	43.8	

	Total	Total %	Quintile					P-value
			Lowest	2nd	3rd	4th	Highest	
			%	%	%	%	%	
NVQ level 5 (Postgraduate degree)	317	3.5	1.1	1.7	5.1	18.9	73.2	
Overseas qualification only	240	2.4	43.4	23.5	20.3	5.7	7.2	
None	1156	11.6	60.1	26.3	8.9	3.6	1.1	
Mother & child health-related variables								
Alcohol consumption during pregnancy								
Never	6606	67.1	21.5	21.1	20.7	20.0	16.7	<0.001
Light	2461	25.7	9.7	14.0	19.6	23.5	33.2	
Moderate	498	5.2	17.8	18.0	18.1	21.6	24.4	
Heavy	204	2.1	22.7	27.1	17.1	15.4	17.7	
Smoking during pregnancy								
Never	6508	66.8	12.8	15.3	20.0	24.7	27.1	<0.001
Quit	1248	12.7	16.7	22.4	24.5	20.3	16.1	
Kept smoking	2048	20.5	36.9	29.8	18.3	9.1	5.9	
Maternal BMI, kg/m^2 at birth								
Underweight (<18.5)	504	5.1	36.2	26.7	13.1	13.6	10.4	<0.001
Normal weight (18.5-24.9)	5907	60.7	15.4	17.4	20.2	21.8	25.2	
Overweight (25-9.9)	1858	18.8	16.6	20.5	20.6	23.6	18.7	
Obese (>29.9)	1536	15.6	26.1	22.6	22.3	16.1	13.0	
Mode of delivery								
Normal	6769	69.3	21.0	20.3	20.2	19.4	19.5	<0.001
Assisted	991	9.7	11.0	16.3	17.9	27.3	27.6	
Planned caesarean	865	8.8	13.2	18.1	24.9	21.3	22.6	
Emergency caesarean	1169	12.2	12.9	16.8	18.9	23.9	27.5	
Breastfeeding								
Never breastfed	3348	33.6	30.0	25.9	20.3	14.4	9.5	<0.001
less than 2 months	2444	24.9	16.7	19.8	21.7	22.3	19.5	
2-4 months	1247	13.1	10.9	15.4	20.2	24.3	29.2	
4-6 months	806	8.4	7.1	10.1	17.7	27.0	38.2	
more than 6 months	1961	20.1	9.9	13.5	19.5	25.4	31.7	
Maternal general health at age 3								
Good health	8108	86.7	15.5	17.6	20.7	22.4	23.8	<0.001
Fair or poor health	1698	13.4	32.2	27.2	18.0	13.3	9.5	
Maternal mental health at age 3								
No or low distress	5568	68.9	10.8	15.6	21.7	24.8	27.2	<0.001
Medium distress	2530	27.7	19.61	22.7	21.2	19.0	17.5	
High distress	274	3.4	45.0	29.0	15.2	6.2	3.7	

	Total	Total %	Quintile					P-value
			Lowest	2nd	3rd	4th	Highest	
			%	%	%	%	%	
Environmental factors								
Exposure to smoking in the home at 9 months								
No	8530	87.3	14.8	17.7	21.0	22.6	23.9	<0.001
Yes	1276	12.7	41.9	29.5	15.1	9.2	4.2	
Damp or condensation in the home at 9 months								
No	8467	86.7	16.2	18.2	20.6	22.1	22.9	<0.001
Yes	1339	13.3	31.8	26.3	18.1	12.8	11.0	
Fume exposure from household heating at 9 months								
No	6421	65.7	17.1	19.0	20.1	21.0	22.8	0.033
Yes	3385	34.3	20.7	19.7	20.6	20.5	18.5	
Rural-urban morphology at 9 months								
Urban	6882	78.7	21.2	19.7	20.1	20.0	19.0	<0.001
Mixed Urban-Rural	812	9.7	9.2	14.9	21.7	25.4	28.8	
Rural	836	11.7	6.5	15.4	20.0	24.0	34.1	

Chi square p-values reported; total in percent and corresponding totals reported; (N=10,260)

3.3.2. Prevalence of asthma-related outcomes

Table 3.5 shows the prevalence of children ever having asthma at ages 3, 5, 7 and 11 by income quintile. At age 3, 11.8% of children had reported ever asthma. This percentage increased to 17.6% at age 11, which is expected given the nature of the question from which this variable was derived. However, once prevalence was stratified by income quintile, there was a general decrease in the prevalence of ever asthma as family income increased– asthma appeared more prevalent in children from families with lower income – and this pattern was consistent across age groups. Asthma prevalence stratified by age and income quintile is depicted graphically in Figure 3.5. There is a general downwards trend of the prevalence of ever asthma as income increases.

Table 3.5: Prevalence of ever asthma by age and income quintile % (n) (N=10,260)

Income quintile	Age 3	Age 5	Age 7	Age 11
Lowest quintile	15.8% (296)	19.4% (319)	18.5% (316)	20.1% (405)
Second quintile	16.5% (315)	18.8% (372)	20.4% (381)	20.2% (458)
Third quintile	12.0% (244)	13.4% (299)	17.0% (349)	17.6% (420)
Fourth quintile	8.3% (172)	11.8% (269)	11.8% (261)	15.2% (392)
Highest quintile	9.2% (189)	10.3% (254)	12.5% (283)	15.7% (416)
Total	11.8% (1,211)	14.3% (1,467)	15.7% (1,611)	17.6% (1,806)

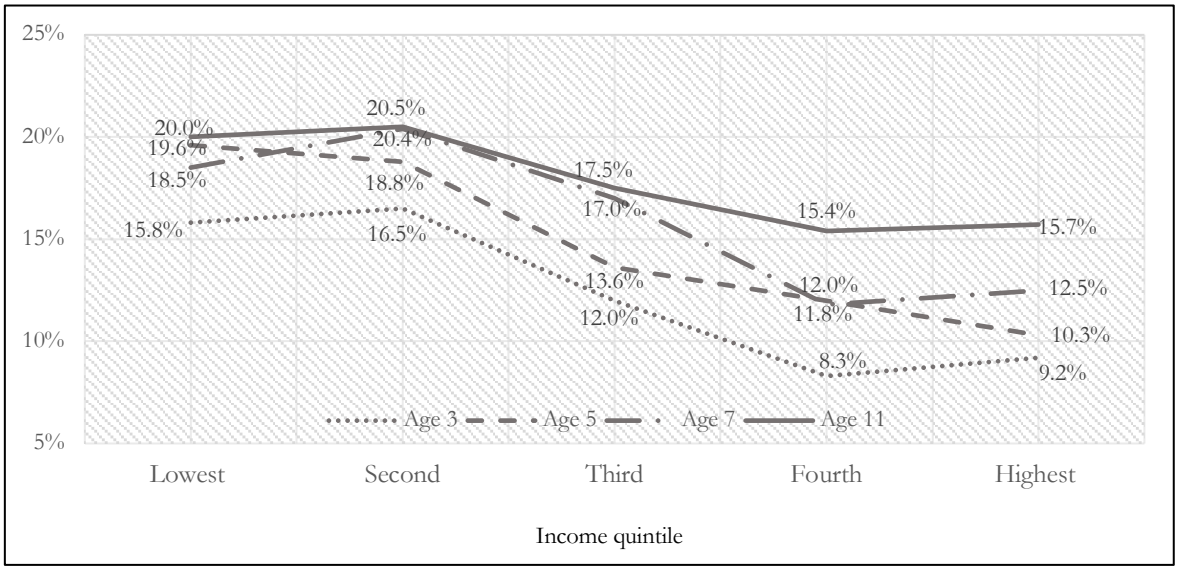


Figure 3.5: Prevalence of ever asthma by age and income quintile

Table 3.6 and Figure 3.6 shows the prevalence of wheezing in the last 12 months for children at ages 3, 5, 7 and 11, by income quintile. The prevalence of wheezing decreased with age from 19.1% at age 3 to around 11% at age 7 and 11. Wheezing in the last 12 months showed a similar income trend as was observed for asthma prevalence – the highest prevalence of wheezing was found in children from families belonging to the lowest income quintiles and there was a lower wheezing prevalence in higher income groups. This trend was especially evident at ages 3 and 5, and less pronounced in later childhood.

Table 3.6: Prevalence of wheezing in the last 12 months by age and income quintile % (n)

Income quintile	Age 3	Age 5	Age 7	Age 11
Lowest quintile	20.8% (357)	19.4% (321)	13.4% (229)	11.1% (225)
Second quintile	22.5% (422)	18.9% (375)	12.8% (242)	13.2% (299)

Third quintile	19.9% (428)	14.6% (326)	12.0% (247)	10.9% (261)
Fourth quintile	17.3% (394)	14.2% (324)	9.6% (214)	10.7% (276)
Highest quintile	16.6% (387)	12.9% (318)	11.2% (254)	11.4% (302)
Total	19.1% (1,960)	15.6% (1,601)	11.7% (1,200)	11.4% (1,170)

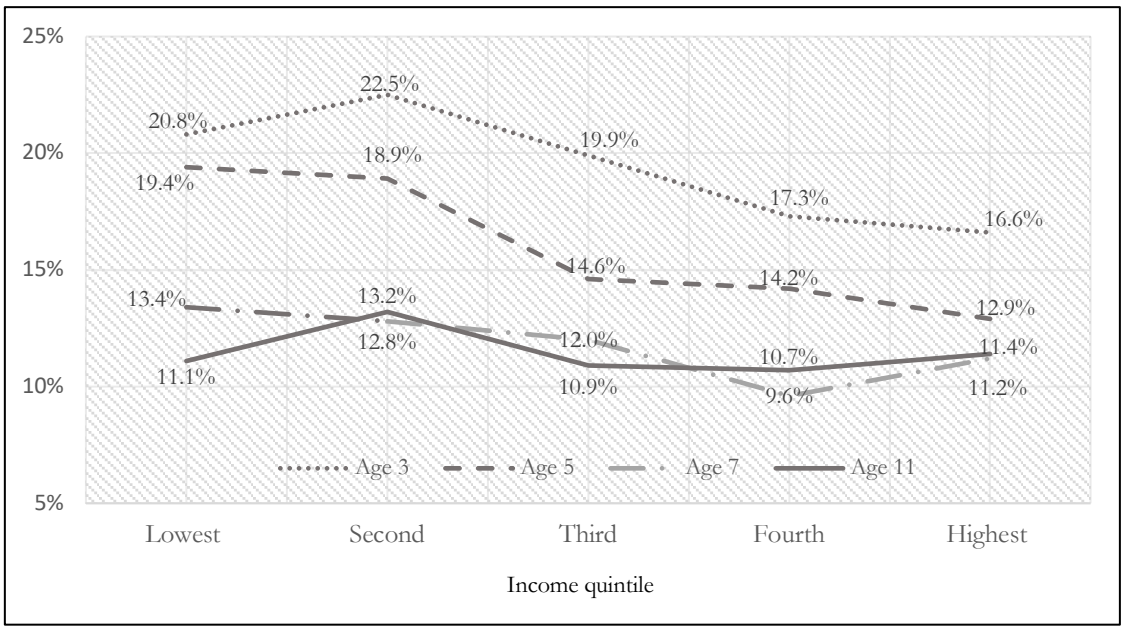


Figure 3.6: The prevalence of wheezing in the last 12 months by age and income quintile

The prevalence of asthma medication use (Table 3.7) was substantially lower in general compared to the prevalence of ever asthma and wheezing in the last 12 months at the same ages. As with ever asthma, the prevalence of asthma medication use increased with age from 3.9% at age 5 to 5.1% at age 11. There was a higher prevalence of asthma medication use in lower income quintiles, this was especially evident at ages 5 and 7 as seen in Figure 3.7. The prevalence of asthma medication use at ages 5 and 7 was 4.9% and 5.3% respectively in the lowest income quintile. These figures were 2.9% and 4.3% respectively in the highest income quintile. Again, the general trend was similar to the distribution of ever asthma and wheezing in the last 12 months where the prevalence in children from low-income families was higher compared to children from high income families.

Table 3.7: Prevalence of asthma medication use by age and income quintile %(n)

Income quintile	Age 5	Age 7	Age 11
Lowest quintile	4.9% (82)	5.3% (90)	4.9% (99)
Second quintile	5.1% (102)	5.2% (98)	6.0% (137)
Third quintile	3.3% (74)	4.2% (86)	5.4% (129)
Fourth quintile	3.5% (81)	3.7% (81)	4.1% (106)
Highest quintile	2.9% (71)	4.3% (98)	5.0% (134)
Total	3.9% (400)	4.5% (462)	5.1% (523)

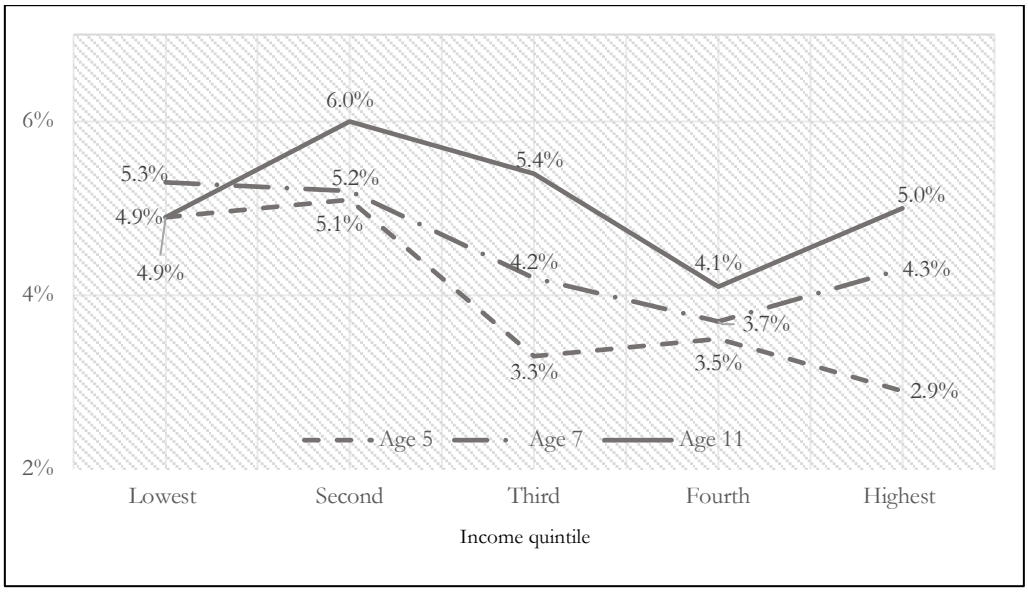


Figure 3.7: The prevalence of asthma medication use by age and income quintile

3.3.3. The association between income and asthma-related outcomes

This section describes empirical results to assess the magnitude and direction of the association between income and asthma-related outcomes at ages 3, 5, 7 and 11. And if this association exists, whether it persists with age.

Marginal effects of income for each of the five model specifications (Table 3.2) are reported in Table 3.8a for ever asthma, Table 3.9a for wheezing in the last 12 months and Table 3.10a for asthma medication use. Tables 3.8b, 3.9b and 3.10b report the corresponding ORs.

3.3.3.1. Ever asthma

At age 3, in the unadjusted model (specification 1), a 1% increase in permanent income was associated with a statistically significant decrease in the probability of ever asthma by 0.05 (marginal effect: -0.05; p-value<0.001; Table 3.8a). On adjusting for a priori variables (specification 2), this decrease in probability remained almost unchanged (marginal effect: -0.05; p-value<0.001). However, after the addition of other socioeconomic variables (specification 3), the decrease in probability was smaller (marginal effect: -0.02; p-value<0.001). When mother and child health-related variables (specification 4) were included in the model the decrease was even less, 1.8% (marginal effect: -0.02; p-value=0.08). Finally, with the inclusion of household and environmental factors (specification 5), the decrease in probability of ever asthma was 0.02 for a 1% increase in income and was statistically significant only at 10% level (marginal effect: -0.02; p-value<0.10). In the corresponding fully adjusted logistic model (specification 5) at age 3, the odds of ever asthma decreased by 0.2% with a 1% increase in income (OR: 0.998; 95% CI: 0.996,1.00; Table 3.8b).

Table 3.8a: The association between income and ever asthma: Marginal effects

Model specification	Age 3	Age 5	Age 7	Age 11
Specification 1	-0.052*** (-0.06, -0.04)	-0.060*** (-0.08, -0.04)	-0.059*** (-0.07, -0.04)	-0.052*** (-0.07, -0.03)
Specification 2	-0.053*** (-0.07, -0.04)	-0.064*** (-0.08, -0.05)	-0.063*** (-0.08, -0.05)	-0.056*** (-0.08, -0.04)
Specification 3	-0.024*** (-0.04, -0.01)	-0.042*** (-0.06, -0.02)	-0.046*** (-0.07, -0.02)	-0.081*** (-0.11, -0.05)
Specification 4	-0.018* (-0.04, 0.001)	-0.036*** (-0.06, -0.01)	-0.038*** (-0.06, -0.02)	-0.075*** (-0.10, -0.05)
Specification 5	-0.017* (-0.04,0.002)	-0.034*** (-0.06, -0.01)	-0.037*** (-0.06, -0.01)	-0.072*** (-0.10, -0.04)

p< 0.10 *; p<0.05 **; p<0.01 ***, 95% Confidence Intervals in parentheses

Table 3.8b: The association between income and ever asthma: Odds ratios

Model specification	Age 3	Age 5	Age 7	Age 11
	0.995***	0.995***	0.996***	0.996***
Specification 1	(0.994,0.996)	(0.994,0.996)	(0.994,0.996)	(0.995,0.997)
	0.995***	0.995***	0.995***	0.996***
Specification 2	(0.993,0.996)	(0.993,0.996)	(0.994,0.996)	(0.995,0.997)
	0.998***	0.996***	0.996***	0.994***
Specification 3	(0.996,0.999)	(0.995,0.998)	(0.995,0.998)	(0.992,0.996)
	0.998*	0.997***	0.997***	0.995***
Specification 4	(0.996,1.000)	(0.995,0.999)	(0.995,0.999)	(0.992,0.997)
	0.998*	0.997***	0.997***	0.995***
Specification 5	(0.996,1.000)	(0.995,0.999)	(0.995,0.999)	(0.993,0.997)

p< 0.10 *; p<0.05 **; p<0.01 ***, 95% Confidence Intervals in parentheses

In the unadjusted logistic model at age 5, a 1% increase in income was associated with a decrease in the probability of ever asthma by 6% (marginal effect: -0.06; p-value<0.001, Table 3.8a). This decrease in probability was 6.4% with the addition of a priori variables and then steadily decreased through specification 3 to 5 with a resulting overall decrease in the probability of a child ever having asthma of 3.4% (marginal effect: -0.03; p-value<0.001). The corresponding OR in the fully adjusted model at age 5 is 0.997 (95% CI: 0.995, 0.999; Table 3.8b). At age 7, the decrease in probability of having asthma associated with a 1% increase in income is 5.9% (marginal effect: -0.06; p-value<0.001). As with the results at age 3 and 5, this association became slightly stronger with the addition of a priori variables (marginal effect: -0.06; p-value<0.001) and is -0.04 in the fully adjusted model (marginal effect: -0.04; p-value<0.001). The associated fully adjusted OR was 0.997 (95% CI: 0.995,0.999). At age 11, a 1% increase in income was associated with a decrease in the probability of ever asthma by 5.2% in the unadjusted model (marginal effect: -0.05; p-value<0.001). Unlike at age 3, 5 and 7, the association between income and ever asthma at age 11 became stronger after the addition of all explanatory variables (marginal effect: 0.07; p-value<0.001). The associated OR was 0.995 (95% CI: 0.993,0.997; Table 3.8b).

In summary, an increase in permanent income was associated with a decrease in the probability that a child had ever had asthma at ages 3, 5, 7 and 11 and the association was statistically significant except at age 3. The marginal effect increased with age: at age 11 it was almost double (marginal effect: -0.07) than that at previous ages (marginal effects of -0.017, -0.034, -0.037 at ages 3,5 and 7 respectively).

3.3.3.2. Wheezing in the last 12 months

In the unadjusted model at age 3 (specification 1), a 1% increase in income was significantly associated with a decrease in the probability of wheezing in the last 12 months by 3.9% (marginal effect: -0.04; p-value<0.001; Table 3.9a). After adjusting for a priori variables (specification 2) the decrease in probability of wheezing in the last 12 months was 4.2% (marginal effect: -0.04; p-value<0.001). In specification 3, with the addition of other socioeconomic factors, the decrease in probability of wheezing in the last 12 months became 2.4% (marginal effect: -0.02; p-value:0.03). Adding mother and child health-related variables to the model (specification 4) further reduced the association and it was no longer statistically significant. Once all explanatory variables were included in the model (specification 5), the association between income and wheezing remained not statistically significant. In terms of ORs, the odds of having wheezing at age 3 reduced by 0.1% with every 1% increase in ln income (adjusted OR: 0.999; 95% CI: 0.998,1.001; Table 3.9b).

Table 3.9a: The association between income and wheezing in the last 12 months: Marginal effects

Model specification	Age 3	Age 5	Age 7	Age 11
Specification 1	-0.039*** (-0.05, -0.02)	-0.042*** (-0.06, -0.03)	-0.023*** (-0.04, -0.01)	-0.014* (-0.03,0.001)
Specification 2	-0.042*** (-0.06, -0.03)	-0.041*** (-0.09, -0.01)	-0.021** (-0.04,0.01)	-0.013 (-0.03,0.003)
Specification 3	-0.024** (-0.05, -0.003)	-0.041*** (-0.12, -0.02)	-0.032*** (-0.05, -0.01)	-0.037*** (-0.06, -0.02)
Specification 4	-0.011 (-0.03, 0.01)	-0.030*** (-0.05, -0.008)	-0.025** (-0.05, -0.003)	-0.026** (-0.05, -0.004)
Specification 5	-0.010 (-0.03, 0.01)	-0.030*** (-0.05, -0.008)	-0.025** (-0.05, -0.003)	-0.025** (-0.05, -0.003)

p< 0.10 *; p<0.05 **; p<0.01 ***, 95% Confidence Intervals in parentheses

Table 3.9b: The association between income and wheezing in the last 12 months: Odds ratios

Model specification	Age 3	Age 5	Age 7	Age 11
	0.998***	0.997***	0.998***	0.999*
Specification 1	(0.997,0.998)	(0.996,0.998)	(0.996,0.999)	(0.997,1.000)
	0.997***	0.997***	0.998**	0.999
Specification 2	(0.996,0.998)	(0.996,0.998)	(0.996,0.999)	(0.997,1.000)
	0.998**	0.997***	0.997***	0.996***
Specification 3	(0.997,1.000)	(0.995,0.999)	(0.995,0.999)	(0.994,0.998)
	0.999	0.998***	0.998**	0.997**
Specification 4	(0.998,1.001)	(0.996,0.999)	(0.995,1.000)	(0.995,1.000)
	0.999	0.998***	0.998**	0.997**
Specification 5	(0.998,1.001)	(0.996,0.999)	(0.995,1.000)	(0.995,1.000)

p< 0.10 *; p<0.05 **; p<0.01 ***, 95% Confidence Intervals in parentheses

At age 5, the association between income and wheezing in the last 12 months in the unadjusted model (specification 1) was of similar magnitude to that at age 3 (marginal effect: -0.04; p-value<0.001; Table 3.8a). With the addition of all explanatory variables (specification 5), the decrease in the probability of having wheezing in the last 12 months associated with a 1% increase in income is 3%, and the odds of wheezing in the last 12 months at age 5 were reduced by 0.2% (marginal effect: -0.03; p-value<0.001); adjusted OR: 0.998; 95% CI:0.996,0.999(Table 3.8b). In the unadjusted model at age 7, the marginal effect of income on wheezing in the last 12 months was -0.023 (marginal effect: -0.02; p-value<0.001). After adjusting for explanatory variables in specification 5, the marginal effect of income increased in magnitude but reduced in statistical significance compared with the unadjusted model (marginal effect: -0.02; p-value:0.05); this corresponded to a decrease in the odds of wheezing in the last 12 months of 0.2% for each 1% increase in income (OR:0.998; 95% CI:0.995,1.00; Table 3.8b). At age 11 the magnitude of the association between income and wheezing in the last 12 months largely decreases compared to younger ages and is only significant at the 10% level (unadjusted marginal effect: -0.01; p-value<0.08; Table 3.9a). In the fully adjusted model (specification 5), a 1% increase in income was associated with a decrease in the probability of wheezing in the last

12 months of 2.5% (marginal effect: -0.03; p-value:0.03); this corresponded to a decrease in odds of 0.3% (adjusted OR: 0.997; 95% CI: 0.995,1.00; Table 3.9b).

3.3.3.3. Asthma medication use

In the unadjusted model at age 5 (specification 1), a 1% increase in income was associated with a decrease in the probability of asthma medication use by 1.5% (marginal effect: -0.02; p-value<0.001; Table 3.10a). With the addition of a priori variables (specification 2), the decrease in probability remained the same and was statistically significant. The addition of socioeconomic variables was associated with an increase in the marginal effect (marginal effect: -0.02; p-value<0.01). After adding mother and child health-related variables (specification 3), the associated marginal effect decreased to -0.02 (p-value< 0.001) and remained the same in the fully adjusted model (Table 3.10a). Finally, a 1% increase in ln income was associated with a decrease in the odds of asthma medication use of 0.8% (OR: 0.995; 95% CI: 0.992,0.998; Table 3.9b).

Table 3.10a: The association between income and asthma medication use: Marginal effects

Model specification	Age 5	Age 7	Age 11
Specification 1	-0.015*** (-0.02, -0.007)	-0.010** (-0.02, -0.001)	-0.007 (-0.02, 0.003)
Specification 2	-0.015*** (-0.02, -0.006)	-0.011** (-0.02, -0.001)	-0.009 (-0.02, 0.002)
Specification 3	-0.023*** (-0.03, -0.01)	-0.023*** (-0.03, -0.01)	-0.026*** (-0.04, -0.01)
Specification 4	-0.018*** (-0.03, -0.01)	-0.020*** (-0.03, -0.01)	-0.023*** (-0.4, -0.01)
Specification 5	-0.018*** (-0.03, -0.01)	-0.021*** (-0.03, -0.01)	-0.024*** (-0.04, -0.02)

p< 0.10 *; p<0.05 **; p<0.01 ***, 95% Confidence Intervals in parentheses

Table 3.10b: The association between income and asthma medication use: Odds ratios

Model specification	Age 5	Age 7	Age 11
Specification 1	0.996*** (0.994,0.998)	0.998** (0.996,1.000)	0.998 (0.996,1.001)
Specification 2	0.996***	0.998**	0.998

	(0.994,0.998)	(0.995,1.000)	(0.996,1.000)
	0.994***	0.995***	0.995***
Specification 3	(0.991,0.997)	(0.992,0.997)	(0.991,0.998)
	0.995***	0.995***	0.995***
Specification 4	(0.992,0.998)	(0.992,0.998)	(0.992,0.998)
	0.995***	0.995***	0.995***
Specification 5	(0.992,0.998)	(0.992,0.998)	(0.992,0.998)

p< 0.10 *; p<0.05 **; p<0.01 ***, 95% Confidence Intervals in parentheses

At age 7, the association between income and asthma medication in the unadjusted model (specification 1) was small but statistically significant (marginal effect: -0.01; p-value:0.04; Table 3.10a). Once additional variables were added to the model, the marginal effect increased, and in the fully adjusted model (specification 5), the marginal effect was -0.02 and statistically significant at the 1% level. The associated decrease in odds of asthma medication use was 0.5% for each 1% increase in income (OR: 0.995; 95% CI: 0.992,0.998; Table 3.10b).

By age 11, the unadjusted association (specification 1) between income and asthma medication use was very small and not statistically significant (Table 3.10a). However, once additional variables were added to the model the marginal effect increased, and in the fully adjusted model (specification 5), a 1% percent increase in income was associated with a statistically significant decrease in the probability of asthma medication use by 2.4% (marginal effect: -0.02; p-value<0.001) and also with a decrease in odds of 0.5% (adjusted OR: 0.995; 95% CI: 0.992,0.998; Table 3.10b).

In summary, there was a statistically significant association between permanent income and asthma medication use for all ages — an increase in permanent income was associated with a decrease in the probability of a child using asthma medication. The magnitude of the association was not as large as it was with ever asthma and wheezing in the last 12 months in both the unadjusted and adjusted models.

3.3.4. Changes in the gradient

In the unadjusted and fully adjusted models the association between income and ever asthma became stronger between age 3 and 11, with an increase in the magnitude of the marginal effect. To compare coefficients between age groups 3, 5, 7 and 11 for all outcomes, Wald tests were performed between models to assess if there are statistical differences in the association of permanent income on asthma-related outcomes across age groups (Cameron and Trivedi, 2010). If estimates are statistically different between ages, one could say that the child health-income gradient changes with age, and if this is patterned in one direction (either increasingly positive or negative) that the gradient in either increasing or decreasing with age. The null hypothesis of the Wald test is that the beta coefficients of income in the models across two successive age-groups are equal:

$$H_0: \beta lny_{age1} = B lny_{age0}$$

$$H_1: \beta lny_{age1} \neq B lny_{age0}$$

where β is the coefficient of the natural logarithm of permanent income for the specified age. A p-value of less than 10% was considered as evidence that the coefficients of income were not equal between specified ages.

P-values for Wald tests based on the fully adjusted models are displayed in Table 3.11a for the outcome ever asthma. Comparing estimated coefficients of income at age 3 to age 5, the p-value was 0.90 meaning that there was no evidence that the association between income and ever asthma was significantly different between age 3 and age 5. The same applies to age 5 and 7 (p-value:0.28). However, comparing age 3, 5 and 7 to age 11, the p-values were less than 0.05 (0.03, 0.02 and 0.04 respectively) therefore the association between income and ever asthma was significantly different between these ages.

Table 3.11a: P-values for differences in the association of income across age: Ever asthma

Wald test p-value	Age 3	Age 5	Age 7	Age 11
Age 3	-	0.904	0.353	0.032**
Age 5		-	0.279	0.015**
Age 7			-	0.042**
Age 11				-

p< 0.10 *; p<0.05 **; p<0.01 ***

Table 3.11b shows the Wald test p-values for outcome wheezing in the last 12 months. There is some evidence that the association between income and wheezing in the last 12 months varied between age 3 and 11 (p-value:0.01), and to a lesser extent between age 5 and 11 (p-value:0.09), but not between the other ages.

Table 3.11b: Differences in the association of income across age: Wheezing in the last 12 months

Wald test p-value	Age 3	Age 5	Age 7	Age 11
Age 3	-	0.213	0.864	0.093*
Age 5		-	0.230	0.013**
Age 7			-	0.115
Age 11				-

p< 0.10 *; p<0.05 **; p<0.01 ***

P-values for Wald tests for the asthma medication models are displayed in Table 3.11c. Again, there was some evidence that the association between income and asthma medication varied between age 5 and 11 (p-value:0.04), but not between the other ages.

Table 3.11c: Differences in the association of income across age: Asthma medication use

Wald test p-value	Age 5	Age 7	Age 11
Age 5	-	0.126	0.035**
Age 7		-	0.361
Age 11			-

p< 0.10 *; p<0.05 **; p<0.01 ***

3.3.5. The association between other factors and asthma-related outcomes

This section outlines the association between all explanatory variables other than family income and asthma-related outcomes. It includes all a priori variables and other confounding variables which remained in the model for specification 5, that is, once fully adjusted for significant socioeconomic factors, mother and child health-related variables and environmental and household variables. These results are displayed in Table 3.13[24].

3.3.5.1. A priori variables

Children from some minority ethnic groups had a lower risk of asthma-related outcomes, although the associations were not always consistent across outcomes or age-groups. For example, in comparison to white children, being of Bangladeshi origin decreased the probability of all asthma-related outcomes although this effect was only statistically significant for ever asthma at age 3 (marginal effect: -0.010), wheezing in the last 12 months at age 11 (marginal effect: -0.09) and asthma medication use at age 5 and 11 (marginal effect: 0.03). Black African children were more likely to have ever had asthma at age 3 (marginal effect: 0.13) but not at other ages, and were less likely to have had wheezing in the last 12 months (marginal effect: 0.06) and to be using asthma medication (marginal effect:-0.03) at age 7 compared to white children. Pakistani children were less likely to be using asthma medication at age 7 (marginal effect:-0.02) or to have ever asthma at age 11 (marginal effect:-0.04).

Across all age groups, male children were significantly more likely to have had asthma, wheezing in the last 12 months and to use asthma medication compared to females. For example, being

[24] Table 3.13 is at the end of this chapter, page 104

male increased the probability of ever asthma and wheezing in the last 12 months by 4-5% at all age-groups and increased the probability of asthma medication use by 1.6-1.9% at age 5-11. At all ages there was a positive and statistically significant association between having a history of asthma in the family and asthma-related outcomes, with an increase in probability of 11-13% across the ages for ever asthma, 7-10% for wheezing in the last 12 months and 3-4% for asthma medication use.

Children with low birthweight (1.5kg<birthweight≤2.5kg) or very low birth weight (<1.5g) have a significantly increased probability of ever asthma at all ages (4-7% for low birthweight and 14-20% for very low birthweight) compared to children with normal birthweight (birthweight: >2.5kg). Similar effects were observed for wheezing and asthma medication use although these effects were not always statistically significant, particularly in the relatively small number of children in the very low birthweight group.

3.3.5.2. Other explanatory variables and potential transmission mechanisms

There may be other behaviours and characteristics linked to parents that may also explain the variation seen in income and asthma-related outcomes. These factors may also independently be determinants of asthma-related outcomes in children. The following section presents estimates of the association between these other explanatory variables and ever asthma, wheezing in the last 12 months and asthma medication use. Because the estimation strategy used a stepwise approach to eliminate variables that were not statistically significant, only those explanatory variables that remained in the final models (i.e. statistically significant at the 10% level) are described in this section, with the exception of mother's education which was included in the models irrespective of statistical significance due to its economic importance. Results are displayed in Table 3.13, at the end of this chapter.

- **Mother's age**

Generally, mother's age appeared to have an inconsistent association with asthma-related outcomes. At age 3, the probability of ever asthma decreased with mother's age, but this effect was only statistically significant for children born to mothers who were 40 or older compared with <20. Positive associations were found between older mothers and wheezing in the last 12 months and asthma medication use in their children at age 5, 7 and 11. In particular, children born to mothers over the age of 20 had an increased probability of wheezing in the last 12 months at age 5 and 7. At age 5, 7 and 11 mothers under the age of 40 were significantly more likely to report asthma medication use in their children compared to mothers under 20.

- **Mother's education**

Mothers' education had no statistically significant association with ever asthma at any age. At age 3, there was no significant association between mother's education and wheezing, however, at ages 5 and 11, children whose mothers had obtained NVQ level 4 and 5 (which corresponds to having a bachelor and postgraduate university degree respectively) were more likely to have wheezing in the last 12 months or report asthma medication use compared to mothers with no qualifications.

- **Housing tenure**

Children from families who lived in homes rented from their local council (as recorded in MCS1) were more likely to report ever asthma and wheezing at age 3 compared to children who were living in owned or mortgaged properties. This association did not hold for families living in privately rented accommodation, but remained statistically significant for ever asthma when children were ages 5 and 7. At age 11 there were no significant associations.

- **Alcohol consumption during pregnancy**

Light and moderate alcohol consumption during pregnancy (compared to no alcohol consumption) were associated with a decrease in the probability of ever asthma (2-3% reduction). Moderate alcohol consumption was also associated with a decrease in the probability of reporting asthma medication use (2-3% reduction) and wheezing in the last 12 months (4% reduction) at ages 5 and 7.

- **Mode of delivery**

Mode of delivery was not significantly associated with asthma medication use but it was associated with ever asthma at age 5 and 11, and wheezing at age 3, 5, and 7. For example, assisted delivery was significantly associated with a higher probability of ever asthma at age 5 and wheezing at age 7 compared to normal delivery. Being born by emergency caesarean section was significantly associated with childhood asthma at age 11 and wheezing at ages 3 and 7. Children born by planned c-section were more likely to have wheezing at age 5 than children born by normal delivery. Overall, the results indicate that children born through delivery modes other than normal delivery – and especially through c-section — were more likely to have poorer asthma-related health outcomes although the patterns were not consistent across outcomes or ages.

- **Breastfeeding**

Children breastfed for longer than 6 months were less likely (4-5% reduction) to have ever had asthma at ages 3, 5 and 7 compared to those who were never breastfed; these children were also significantly less likely to have wheezing at 3 or 5 (4% reduction). Breastfeeding for 2-4 or 4-6 months was also associated with a decrease in the probability of ever asthma and wheezing in the last 12 months but the effect was generally smaller and not always statistically significant. Breastfeeding was not significantly associated with any asthma outcome at age 11.

- **Mother's general health and mental health**

Children whose mothers have fair or poor general health were more likely to have ever asthma, wheezing or take asthma medication at age 5, 7 and 11 compared to mothers with good or excellent general health, with increases in probability of these outcomes ranging between 2.1 and 4.5%. Children whose mothers had poor mental health (high distress levels as measured using the Kessler scale) were more likely to have ever asthma at age 3 compared to those whose mother had good mental health (low distress levels). Children whose mothers had fair and poor mental health were also more likely to have wheezing at all ages.

- **Condensation or damp in the household**

With respect to the household environment, condensation or damp reported in the home did not appear to have a strong association with asthma-related outcomes. The only significant association was present at age 11 when children who lived in homes with damp or condensation were more likely to have ever had asthma at age 11.

- **Exposure to fumes from heating**

Children who were exposed to fumes from household heating such as from gas and wood fires when they were 9 months old were more likely to have poor asthma-related outcomes. At age 3, these children were more likely to have asthma, wheezing and be using asthma medication. They were also more likely to have some asthma-related outcomes at age 5 (ever asthma and asthma medication), age 7 (ever asthma); and at age 11 (wheezing in the last 12 months) compared to those who were not exposed to fumes in the home.

- **Rural-urban morphology**

Whether a child lived in a rural, urban or in a rural-urban mix appeared not to be associated with asthma-related outcomes at any age. The only significant association was found in children who lived in a mixed urban and rural area or in a rural area at MCS 1, they were significantly less likely to have wheezing in the last 12 months at age 3 compared to those living in urban areas.

3.3.6. Sensitivity analysis results

Results for lagged income showed evidence of an income-child health gradient with outcome measures ever asthma and asthma medication use, albeit to a lesser extent. In fully adjusted results for ever asthma at age 3 (specification 5), a 1% increase in income was associated with a decrease in the probability of a child having asthma by 0.018 (marginal effect:-0.018; p-value:0.039) compared to -0.017 with permanent income. At age 7 the marginal effect was -0.028 (p-value:0.001) compared to -0.037 with permanent income and by age 11, -0.03 (p-value:0.001) compared to -0.072 in the case of permanent income. When the outcome was wheezing in the last 12 months, at age 3 (specification 5) there was a statistically significant association between lagged income and wheezing (marginal effect: -0.028; p-value:0.011) compared to permanent income results (marginal effect -0.010; p-value:0.14). By age 11 the marginal effect was -0.013 (p-value:0.275) compared to permanent income results (marginal effect: -0.025; p-value:0.05). When asthma medication use is the outcome measure, there was statistically significant association between lagged family income and asthma medication use at age 5 (marginal effect: -0.008; p-value:0.01) compared results with permanent income (marginal effect:-0.018; p-value:0.002). At age 7 the magnitude of the association decreased to -0.009 (p-value: 0.037) compared to permanent income results (marginal effect: -0.021; p-value: 0.01) and at age 11 was -0.013 (p-value:0.019) compared to results with permanent income (marginal effect: -0.024; p-value:0.01) (Tables 3.12a, 3.12b and 3.12c display marginal effects of income

across age for each asthma-related outcome). As with the case of permanent income, there was also a significant increase in the strength of association between income and asthma-related outcomes between age 3 and 11. Full results of the sensitivity analyses can be found in Appendix B-2 and B-3.

Table 3.12a: The association between lagged income and ever asthma: Marginal effects

Model specification	Age 3	Age 5	Age 7	Age 11
Specification 1	-0.044*** (-0.06, -0.03)	-0.034*** (-0.04, -0.02)	-0.046*** (-0.06, -0.03)	-0.035*** (-0.05, -0.02)
Specification 1 (perm)	-0.052***	-0.060***	-0.059***	-0.052***
Specification 5	-0.018** (-0.035- -0.001)	-0.006 (-0.020-0.007)	-0.028*** (-0.044- -0.012)	-0.031*** (-0.049- -0.012)
Specification 5 (perm)	-0.017*	-0.034***	-0.037***	-.072***

p< 0.10 *; p<0.05 **; p<0.01 ***, 95% Confidence Intervals in parentheses. Results for permanent income (perm) shown for comparison.

Table 3.12b: The association between lagged income and wheezing in the last 12 months: Marginal effects

Model specification	Age 3	Age 5	Age 7	Age 11
Specification 1	-0.041*** (-0.055, -0.027)	-0.027*** (-0.038, -0.016)	-0.015*** (-0.026, -0.004)	-0.011* (-0.023, -0.001)
Specification 1 (perm)	-0.039***	-0.042***	-0.023***	-0.014*
Specification 5	-0.028** (-0.05, -0.006)	-0.010 (-0.024,0.004)	-0.013* (-0.027,0.001)	-0.008 (-0.023,0.006)
Specification 5 (perm)	-0.010	-0.03***	-0.025**	-0.025**

p< 0.10 *; p<0.05 **; p<0.01 ***, 95% Confidence Intervals in parentheses. Results for permanent income (perm) shown for comparison.

Table 3.12c: The association between lagged income and asthma medication use: Marginal effects

Model specification	Age 5	Age 7	Age 11
Specification 1	-0.010*** (-0.015, -0.005)	-0.005 (-0.012, 0.001)	-0.007* (-0.015, 0.001)
Specification 1 (perm)	-0.015***	-0.01**	-0.007
Specification 5	-0.008*** (-0.015, 0.002)	-0.009** (-0.017, 0.001)	-0.013** (-0.023, 0.002)
Specification 5 (perm)	-0.018***	-0.021***	-0.024***

p< 0.10 *; p<0.05 **; p<0.01 ***, 95% Confidence Intervals reported in parentheses. Results for permanent income (perm) shown for comparison.

3.4. Discussion

3.4.1. Summary of results

This chapter has examined the association between family income and three asthma-related outcomes at age 3, 5, 7 and 11 years. For all outcomes, the probability of the outcome decreased with increasing family income, even after adjusting for many other variables. In the fully adjusted model, the decrease in the probability of ever asthma was 1.7% at age 3, rising to 7.2% at age 11, and there was evidence that the coefficient increased as the children became older. In the fully adjusted models for wheezing in the last 12 months and asthma medication use, the decrease in probabilities at ages 5-11 were about 3% and 2% respectively. When using lagged income rather than permanent income, the coefficients for income were somewhat smaller although they were still statistically significant. Changes in the child health-income gradient were similar to the changes found with permanent income.

3.4.2. Age-specific prevalence of asthma-related outcomes

In singleton children in the MCS, the overall prevalence of ever asthma increased from 11.8% at age 3 to 17.6% at age 11. Over a longer time-frame, it is more likely that children will have. experienced some asthmatic symptoms. My findings are consistent with other studies that have shown that at preschool age, roughly 3 to 5 years old, only 10-15% of children will have had a true diagnosis of asthma (van Aalderen, 2012). In relation to income, I also found that the prevalence of ever asthma within each age group was lower in children in the highest income quintile and the highest prevalence being found in the lowest income quintile (Table 3.1).

The prevalence of reported wheezing in the last 12 months decreased from 19.1% at age 3 to 11.4% at age11. Literature has shown that up to 50% of children will have had at least one

episode of wheezing by the age of 5 (Subbarao, 2009). Wheezing tends to be associated with viral infections and environmental stimuli so it is expected that a higher number of children are reported as having had wheezing in early childhood when they are more susceptible to infections, that resolve later in childhood, typically by age 6 (Hossny et al., 2017). Wheezing is also very common in younger children; it is estimated that as much as one third of young children in the UK have wheezing attributable to their airways not being fully developed (Iqbal, 2012). As is the case with ever asthma, wheezing in MCS children is more prevalent in children from low-income families compared with high income families.

In the case of asthma medication use, the prevalence increased with age from 3.9% at age 5 to 5.1% at age 11, but at all ages it was substantially lower than for ever asthma and wheezing in the last 12 months. This increase was expected because as children move from early childhood into later childhood it is likely that, if they have asthmatic symptoms that persist beyond early childhood wheezing, they would eventually be diagnosed with asthma and start a specific asthma treatment. At all ages, the prevalence of asthma medication was higher in children from low-income families compared with high-income families.

The results from regression modelling in this chapter supported these descriptive results on the association between income and asthma-related outcomes. Statistically significant associations between income and asthma-related outcomes were consistently found for different outcomes and across different age-groups, supporting the hypothesis that income is a significant determinant of these outcomes. These results are discussed in the following section.

3.4.3. Income as a determinant of asthma-related outcomes in children

There is a wide body of research from around the globe that supports the notion that children who grow up in poor families experience a large range of adverse outcomes, including poor health outcomes. Policy makers have used this evidence to justify increased welfare to low-income families and their children. However, much of the research mainly focuses on general health outcomes in children. The analyses in this chapter expand on this research by providing evidence using the specific case of asthma-related outcomes in children in the UK.

Overall results from the cross-sectional analysis show that there are statistically significant relationships between low income and ever asthma, wheezing in the last 12 months and asthma medication use. The results are comparable to published results from other MCS analysis of the association between income and child respiratory health outcomes. In a previous study using the MCS and the same measure of income (based on interval regression now provided by the CLS) and same health outcomes, Violato et al (2009) found the unadjusted marginal effect of permanent income on ever asthma at age 3 was found to be -0.041(p-value<0.01) compared to 0.052 (p-value<0.01) in my analysis, and the fully adjusted marginal effect was -0.014 (0.05<p-value<0.10) compared to -0.017 (p-value:0.003) in my analysis. The fully adjusted models included a similar but not identical set of explanatory variables which might account for some of the differences in values. In the same study, the marginal effect at age 5 was -0.043 (p-value<0.01) and -0.014 (0.05<p-value<0.10) in unadjusted and fully adjusted models respectively compared to -0.060 (p-value<0.01) and 0.034 (p-value<0.01) in my results; coefficients are larger but again the set of explanatory variables was not identical. For wheezing the results were very similar, at age 3 marginal effects of -0.040 (p-value<0.01) and -0.012 (p-value>0.10) in unadjusted and adjusted models compared to -0.039 (p-value<0.01) and -0.010

(p-value>0.01) in my results. At age 5 these were -0.040 (p-value<0.10) and -0.025 (0.05<p-value<0.1) compared to -0.042 (p-value<0.010) and -0.030 (p-value<0.010) in my analysis. Differences in coefficients in adjusted models are expected due to inconsistencies between the groups of explanatory variables included in the studies. Other explanations as to why these differences arise are: firstly, the income variable used in my research was a derived variable provided in the MCS data, whereas in the Violato et al (2009) study income was calculated using interval regression and income bands. Secondly, the study population in my analysis only included children who were present in all five surveys, whereas the Violato et al (2009) study did not use this inclusion criteria.

Other studies that have looked specifically at the association between income and asthma or asthma-related outcomes support the findings in this chapter. Propper et al (2006), found that a 1% increase in income (measured as the ln average of income measured at 33 and 47 months) statistically significantly reduced the odds of persistent wheeze (between birth and 42 months) to 0.87 (standard error: 0.07) and asthma at month 81 to odds of 0.81 (standard error: 0.06) showing low income to be an important determinant of wheezing.

There is also some evidence from my results to support the hypothesis that the association between low-income and asthma-related outcomes tends to become stronger with age i.e. strengthening of the child health- family income gradient. For outcome ever asthma, there are statistically significant differences between the marginal effects of income at ages 3, 5, 7 compared to 11, demonstrating that the effect of income is stronger at age 11. My results showed that comparing results between ages 3 and 11 for ever asthma the p-value was 0.03, for ages 5 and 11 (p-value: 0.015) and ages 7 and 11 (p-value:0.042) (Table 3.11a). For wheezing in the last 12 months there is no evidence of the gradient strengthening and for asthma medication use, there was a significant difference between the age 5 and age 11 effects, but there are only three time periods thus the pattern of strengthening is not clear. An explanation for the stronger

gradient seen at age 11 could be that children in low income are more likely to suffer illness at older ages which includes both chronic and new diseases. Previous studies in the UK (Currie and Stabile, 2003, Propper et al., 2007, Doyle et al., 2007, Violato et al., 2009 and Kuehnle, 2014), the latter two studies also using the MCS, were inconclusive on whether the gradient becomes stronger with age. However, these studies did not include follow-up until age 11 as was the case in my analysis.

Overall, the results of the sensitivity analysis show that the size of the effect of lagged income was smaller than in the case of permanent income for all outcomes and across all ages. These findings are in line with previous studies and suggest that health investments are based on long run average income rather than on when the income was received (Case et al., 2002, Currie et al., 2007, Violato et al., 2009, Kuehnle 2014).

In summary, the results from my cross-sectional analysis add to the limited evidence that there is a significant association between low income and adverse asthma-related outcomes in children in the UK. Children from poor families are worse off in terms of their health – an issue which needs to be addressed urgently. These findings are also particularly important given that the current trend in the UK is increasing household costs (which include the cost of raising a child) with unmatched increases in income levels (Hirsch, 2019). Therefore, there is a need to assist low- income families by means of welfare, for example through improved child welfare (more detail in Chapter 7).

3.4.4. Other risk factors for asthma-related outcomes

3.4.4.1. A priori factors

In addition to the association between income, the results show statistically significant associations between a priori confounders and asthma-related outcomes. The general finding is

that asthma and wheezing are more prevalent in males during infancy. Gender differences in asthma and wheezing have been researched extensively and research has shown that this reverses at some point during adolescence (Propper et al., 2007; Arathimos et al., 2017). It is likely that analysis of later MCS data when children are older would reveal the same reversal which will see asthma-related outcomes more prevalent in females.

The results also show a statistically significant association between a family history of asthma and adverse asthma-related outcomes across all ages. This is in accordance with previous findings that find that heritability plays an important role in determining the presence of asthma and wheezing in children (Civelek et al., 2010; Lim et al., 2010; Ullemar et al., 2015).

In terms of birthweight, my results show that low and very low birthweight are associated with adverse asthma-related outcomes. These results are in line with the literature on the risks associated with low birthweight and prematurity. This highlights the importance of perinatal child health in determining respiratory health later in childhood (Villamor et al., 2009). However, it is unclear whether low birthweight itself is a risk factor for asthma or whether it is instead indicative of sub-optimal intrauterine conditions. Regardless of the cause, the association of low birthweight on asthma-related outcomes still holds even if exact mechanisms underlying the association are still not well understood.

3.4.4.2. Other socioeconomic factors

The results show that children with more highly educated mothers are more likely to be using asthma medication. According to previous literature, more educated mothers may have better knowledge about health and the health care needs of their children which might have a bearing on adherence rates and consistent medication use (Chen and Li, 2009). This may also impact health seeking behaviour, as increased health knowledge may make mothers more likely to take children with asthma-related symptoms to a health care professional, who may then diagnose

and prescribe asthma medication. In considering mother's education as a measure of socioeconomic status, previous literature has also indicated that low socioeconomic status is associated with poor medication adherence. This could be due to the out-of-pocket payments that need to be made to access health care facilities and pharmacies as well as repeat prescription payments, although prescriptions for children are free in the UK (El-Rachidi et al., 2017)

With respect to mother's age at birth of the cohort child, children born to mothers over the age of 20 are more likely to use asthma medication compared to those born to mothers below the age of 20. It is likely that mothers over 20 have acquired more education including health education and have more experience with the management of their child's health compared to younger and less experienced mothers. Although there is not an abundance of literature on the association of maternal age at birth and child health outcomes, studies in the UK and other countries such as Canada and Japan have found that older maternal age is significantly associated with improved child health outcomes, and in the case of asthma specifically, that asthma is significantly less common in children with older mothers (Laerum et al., 2007; Kato et al, 2017).

With respect to housing, the results demonstrate that for a child living in council rented housing, there is an increased probability of ever asthma, wheezing in the last 12 months and asthma medication use compared to children whose parents owned their homes. Previous studies have shown that children who live in social housing or council housing experience greater levels of disadvantage, including poorer health outcomes and this relationship also holds for asthma (Violato et al., 2009; Kuehnle, 2014; Nasim, 2015). In addition to socioeconomic status, housing tenure could also be an indicator of housing quality or environmental condition where rented homes (council or privately rented) may have higher levels of damp compared to owned homes and may generally be in more polluted areas, thus having direct effects on asthma-related outcomes (Kuehnle, 2014).

3.4.4.3. Mother and child-health related factors

Overall, breastfeeding for more than six months significantly decreases the probability of ever asthma. These findings are in line with the literature that support the notion that limited or no breastfeeding has a negative effect on childhood asthma and wheezing (Propper et al., 2006; Violato et al., 2009; Kuehnle, 2014). Generally, the protective effect of breastfeeding on asthma and wheezing in particular has had mixed findings in epidemiological literature (Lodge et al., 2014). These inconclusive findings are likely to be attributable to the differences in the way breastfeeding is recorded e.g. duration of breastfeeding, whether breastfeeding was exclusive and to the heterogeneity in reporting and measuring asthma-related outcomes. However, the overall benefits of breastfeeding beyond asthma and wheezing are undisputed and exclusive breastfeeding is very much encouraged for the first 6 months of infancy to achieve optimal health (WHO, 2001; RCPCH, 2019).

Significant negative associations found between moderate alcohol consumption during pregnancy and all three outcomes at ages 5 and 7 need to be interpreted with caution; generally they support evidence that is inconclusive regarding the negative impact of moderate alcohol consumption during pregnancy on child asthma (Magnus et al., 2014).

Assisted deliveries, planned and emergency c-sections are associated with an increased probability of all asthma-related outcomes. My results agree with recent findings that babies born by c-section are more likely to suffer from asthma and wheezing in childhood (Magnus et al., 2011; Keag et al., 2018). One potential explanation for the association is the 'hygiene hypothesis', which implies that there may be certain immunological properties gained from normal delivery from exposure to microbes in vaginal flora which attribute to the development of the immune system. Therefore, babies born by c-section may have a less developed immune system making them more susceptible to asthma-related outcomes however, this theory is

disputed by others (Braun-Fahrlander et al., 2002; Magnus et al., 2011; Azad et al., 2013). Another explanation is that children born by c-section in general have a shorter gestational age and are prone to more neonatal respiratory health problems – both indicators of the importance of an optimal intrauterine environment (including optimal gestational age) in determining the development of asthma and wheezing (Magnus et al., 2011).

Finally, my results provide strong evidence that poor general health and poor mental health in mothers increased the probability of a child having adverse asthma-related outcomes compared to children whose mothers had good general and mental health. This is in agreement with previous findings that a child's health is strongly influenced by the health of their mothers through a number of potential pathways. It might be from an inherited susceptibility to disease in general or a less healthy uterine environment, which then translates to poor health in early childhood (Kuehnle, 2014). The main explanation offered in the literature is that less healthy mothers are less able to deliver a good level of care to their children as their level of interaction may be limited due to time spent recovering from illness (Case et al., 2002). Another explanation is that there may be common but unmeasured environmental factors resulting in a correlation between mother and child health levels (Case et al., 2002; Ghaempanah et al., 2013; Kuehnle, 2014).

3.4.5. Strengths and limitations

The strengths of the study are largely linked to the strengths of the MCS itself and availability of a rich data set. The large population-based sample size of the MCS which allows for a higher degree of generalisability of results to the wider UK population. Additionally, the MCS provides representative data across socioeconomic groups in England, Northern Ireland, Scotland and Wales which allows results to be applicable to the current UK population and socioeconomic situation. The MCS provides a large number of variables which are consistently measured at

every age, thus allowing adjustment for a large number of potential confounders. The analysis operationalised income in different ways (permanent income and lagged income) which allows for a robustness check of the results – in this case, permanent income had a larger association compared to lagged income, but the results were still comparable.

Three different measures of asthma-related outcomes were explored, of which two (ever asthma and wheezing in the last 12 months) are validated questions taken from the ISAAC questionnaire and have been widely used in the literature on asthma and wheezing, again making them good for comparison across the literature (Connelly and Platt, 2014). Having different measures of asthma-related outcomes is also useful as a robustness check and indeed results show that the general theme of low income and adverse asthma outcomes holds regardless of outcomes used.

Some limitations are that the outcome variables ever asthma and wheezing in the last 12 months are subjective parent-reported measures of child health which require the parents to recall over the past 12 months or longer, so I cannot rule out reporting biases. Ideally patient records combined with physiological data would provide more accurate measures of asthma-related outcomes. Asthma medication use is not subjective as it is based on current, specific asthma medications, but it still requires reliable recall.

Asthma-related outcomes in my analyses are limited to dichotomous measures and these may not reflect the heterogeneity of asthma and wheezing. To date, asthma has not been uniformly defined and its association with wheezing is also not clear. Binary outcomes therefore do not contain or reflect the full spectrum of asthma-related outcomes which may differ in duration as well as severity (Deliu et al., 2017). However, the phenotype analysis and the panel data analysis in subsequent chapters provide a way to account for the persistence and duration of asthma-related outcomes over time and help overcome the limitation of using a dichotomous measure at a single point in time.

Attrition rates and subsequent missing data pose a potential issue with the analysis – from the 18,553 families included at the age 9 months survey (MCS 1), only 10,260 families were used in the analysis as these children were present in all of the surveys (9 months, ages 3, 5, 7 and 11 years). If there were characteristics that were correlated with being lost to follow-up then the results could arguably be biased. Fortunately, the sample weights included in the analysis accounted for attrition bias by adjusting for many potential variables that are associated with attrition at each survey age. However, if attrition is associated with unobserved and thus unrecorded characteristics (and thus not included in sample weight calculation) then there is added potential for biased results. Most of the missing data in the study was due to attrition rather than item non-response, hence it was not necessary to employ other techniques to deal with missing data.

3.5. Conclusion

The results provide new evidence, using a broader range of data of children from the age of 3 into early adolescence at age 11, that low income has a significant association with adverse asthma-related outcomes. The numerous explanatory variables included in the analysis also shed light on the channels through which low family income translates into poor asthma-related outcomes in children. These findings are based on repeated cross-sectional analysis at four different age-groups and do not exploit the longitudinal nature of the data. In Chapter 4, I create phenotypes based on how the asthma-related symptoms change over time and then look at the association between family income and these phenotypes and in Chapter 5, I use longitudinal data analysis to explore whether the association between family income and asthma-related outcomes changes over time.

Table 3.13: The association between income and other factors: Marginal effects

	Ever asthma				Wheezing in the last 12 months				Asthma medication use		
	Age 3	**Age 5**	**Age 7**	**Age 11**	**Age 3**	**Age 5**	**Age 7**	**Age 11**	**Age 5**	**Age 7**	**Age 11**
Ln income	-0.017*	0.034***	0.037***	0.072***	-0.010	-0.030***	-0.025**	-0.025**	-0.018***	-0.021***	-0.024***
Ethnicity											
White	ref										
Mixed	0.051	0.033	0.004	0.020	0.002	0.046	0.032	-0.007	0.022	0.020	-0.006
Indian	-0.008	-0.015	-0.003	-0.009	-0.014	0.054	-0.007	0.012	-0.013	0.013	0.024
Pakistani	-0.032	-0.006	-0.001	-0.038**	0.012	0.037	-0.003	-0.009	-0.006	-0.019**	-0.012
Bangladeshi	-0.104***	-0.019	-0.017	-0.041	-0.048	-0.033	-0.001	-0.088***	-0.033***	-0.020	-0.028**
Black	0.073	0.081	0.045	-0.040	0.078	0.050	0.034	0.006	-0.013	0.015	0.018
Black African	0.125**	0.004	-0.040	-0.035	-0.016	-0.045	-0.058**	0.006	0.004	-0.032***	0.005
Other	0.021	-0.033	-0.018	-0.069***	-0.114***	-0.081***	-0.006	-0.043*	-0.013	-0.020**	-0.012
Child sex											
Male	0.041***	0.047***	0.051***	0.048***	0.048***	0.050***	0.046***	0.043***	0.016***	0.016***	0.019***
Birthweight											
Normal	ref										
Low	0.044**	0.066***	0.046**	0.071***	0.054**	0.056**	0.036*	0.002	0.053***	0.014	0.002
Very low	0.137**	0.165***	0.195***	0.164**	0.164**	0.115*	0.050	0.017	0.054	0.077	0.070
Family History of asthma	0.114***	0.110***	0.126***	0.123***	0.104***	0.081***	0.077***	0.072***	0.029***	0.043***	0.036***
Number of siblings											
No siblings	ref										
1 sibling	0.013	0.001	-0.007	-0.010	0.026**	-0.005	-0.006	-0.012	-0.007	-0.006	-0.016**
2 siblings	0.015	-0.019	-0.019	-0.028*	-0.011	-0.008	-0.008	-0.023**	-0.014*	-0.025***	-0.034***
3 or more	0.040**	-0.021	-0.018	-0.032	0.017	-0.008	0.001	-0.010	-0.003	-0.007	-0.021**
Mother's education											

	Ever asthma				Wheezing in the last 12 months				Asthma medication use		
No qualification	ref										
NVQ 1	-0.002	-0.004	-0.022	-0.004	-0.012	0.005	-0.015	0.029*	-0.002	0.026***	0.003
NVQ2	0.000	-0.007	-0.006	-0.006	-0.006	0.022	-0.010	0.019	-0.002	0.013**	0.004
NVQ3	-0.017	-0.014	-0.023	-0.009	-0.024	0.000	-0.023	-0.003	-0.007	0.010	-0.007
NVQ4	0.019	-0.010	0.010	0.013	0.003	0.029*	0.000	0.037**	0.008	0.030***	0.015
NVQ5	0.027	0.005	0.017	0.038	0.013	0.051*	0.023	0.056**	0.041*	0.041**	0.034*
Overseas qualification	0.010	0.003	0.011	0.021	0.015	0.019	-0.001	0.028	0.013	0.014	0.008
Mother's age											
<20	ref										
20-24	0.011	-	-	0.036*	0.028	0.021	0.033**	0.013	0.023***	0.031***	0.008
25-29	-0.017	-	-	0.036*	0.017	0.032*	0.045***	0.006	0.023***	0.029***	0.025**
30-34	-0.027	-	-	0.038*	0.018	0.033*	0.045***	0.018	0.026***	0.032***	0.022**
35-39	-0.030	-	-	0.058**	0.002	0.042**	0.062***	0.027	0.025***	0.037***	0.025**
>=40	-0.064**	-	-	0.016	0.075*	0.088**	0.080***	0.043	0.029*	0.005	0.027
Housing tenure											
Mortgage /owned	ref										
Council rented	0.032**	0.029**	0.043***	-	0.039*	0.021	0.025	-	-	-	-
Rented	0.017	0.022	0.021	-	0.012	0.013	-0.010	-	-	-	-
Breastfeeding											
Never	ref										
<2 months	-0.006	-0.008	-0.005	-	-0.021	-0.007	-0.008	0.001	0.004	-	-
2-4 months	-0.026**	-0.027**	-0.023*	-	-0.04**	-0.022	-0.025*	-0.011	-0.003	-	-
4-6 months	-0.029*	-0.021	-0.001	-	-0.043*	-0.021	-0.007	0.029	-0.012	-	-
>6 months	-0.054***	-0.045***	-0.036***	-	-0.041***	-0.042***	-0.013	0.012	-0.013*	-	-
Mode of delivery											
Normal	ref										
Assisted	-	0.030**	-	0.017	0.025	0.008	0.023*	-	-	-	-

	Ever asthma				Wheezing in the last 12 months				Asthma medication use		
Planned csection	-	0.020	-	-0.001	0.027	0.036**	0.009	-	-	-	-
Emergency csection	-	0.015	-	0.039***	0.04***	0.016	0.028**	-	-	-	-
Mother's general health											
Excellent or	ref										
Fair or poor	-	0.026**	0.045***	0.032**	-	0.026*	0.033**	0.034***	0.027***	0.024***	0.021**
Mother's mental health											
Low distress	ref										
Medium	0.015	-	-	-	0.031***	0.043***	0.019**	0.000	0.005	-	-
High distress	0.048**	-	-	-	0.11***	0.041	0.010	0.034*	0.019	-	-
Alcohol consumption											
None	ref										
Light	-	-0.024***	-0.022**	-	-	-0.013	-0.012	-	0.003	-0.009	-
Moderate	-	-0.034**	-0.034*	-	-	-0.042***	-0.040***	-	-0.021***	-0.029***	-
Heavy	-	-0.005	0.007	-	-	0.022	-0.035	-	-0.007	-0.018	-
Exposure to fumes	0.012*	0.023***	0.020**	-	0.018*	-	-	0.019**	0.010**	-	-
Damp	-	-	-	0.031**	-	-	-	-	-	-	-
Rural/Urban morphology											
Urban	ref										
Mixed	-	-	-	-	-0.029*	-	-	-	-	-	-
Rural	-	-	-	-	-0.024*	-	-	-	-	-	-

p< 0.10 *; p<0.05 **; p<0.01 ***; Where variables were dropped from fully adjusted models, all categories for a given variable appear as blank for that specific age and outcome (-). Reference categories are represented by "ref".

4.Income and wheezing phenotypes

4.1. Phenotypes in relation to asthma aetiology

A phenotype can be described as a characteristic or set of characteristics that arise from the interaction of genes and environmental exposures that can be directly observed and measured (Wenzel, 2012; Deliu et al., 2017). In terms of the link between phenotype and asthma-related outcomes, the latter which is the focus of this thesis, wheezing is a major clinical symptom of asthma and therefore wheezing phenotypes have been used extensively to characterise asthma. Wheezing phenotypes were first defined by Martinez et al (1995) using data from the Tuscon Children's Respiratory Study (commonly referred to as the Tuscon study). The study enrolled children born in the US between 1980 and 1984 and collected data using questionnaires completed by mothers and paediatrician records of episodes of lower respiratory tract infections at birth, ages 2 and 6 years. Using these data, children were assigned to one of four categories according to their history of wheezing and lower respiratory tract infections. Categories assigned were 'no wheezing' (those who had no records of lower respiratory tract illness with wheezing in the first 3 years and who reported no wheezing at age 6); 'transient early' (children who had had at least one lower respiratory tract illness with wheezing during the first 3 years of life, but no wheezing at age 6), 'late onset' (children who had no lower respiratory illness with wheezing in the first 3 years, but who reported having wheezing at age 6); and 'persistent' (children who had at least one lower respiratory tract illness with wheezing in the first 3 years and wheezing at age 6) (Martinez et al., 1995). According to these phenotypes, of the 826 children included in the study, 51% had 'no wheezing', 20% had 'transient early' wheezing, 15% had 'late onset' wheezing and 14% had 'persistent' wheezing (Figure 4.1) (Martinez et al., 1995).

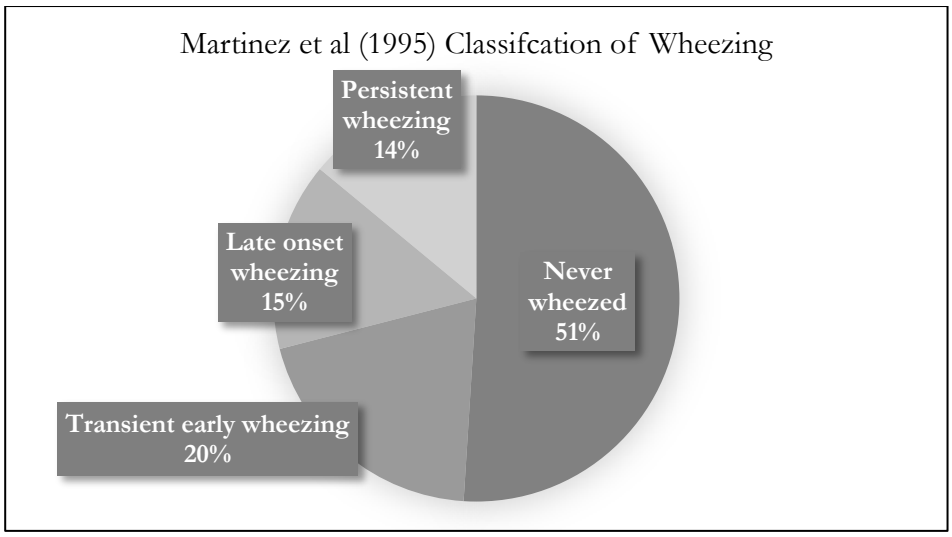


Figure 4.1. Wheezing phenotypes classified by Martinez et al (1995)

Wheezing phenotypes have specifically been chosen as the health outcome for the analysis in this chapter, as these phenotypes take into account the heterogeneous nature of asthma and how it presents (Deliu et al, 2017). These phenotypes are characterised by distinct wheezing patterns identifiable in childhood, such as those described in the Tuscon study. Phenotypes may also be influenced by age, gender, genetic background and environmental exposures (Deliu et al., 2017; Brew et al., 2019). Economic and epidemiological studies that have accounted for heterogeneity in asthma, have largely done so by exploiting variation in time of onset, duration of symptoms and recurrence of wheezing over time, which is then used to define phenotypes (Martinez et al., 1995; Taylor-Robinson et al., 2015; Deliu et al., 2017).

Characterising wheezing into phenotypes in this way, allows for some of the limitations of the analysis in Chapter 3 to be addressed by developing a more varied categorical health outcome, compared to the dichotomous asthma-related outcomes used previously. Wheezing phenotypes allow for the inclusion of a number of categories of wheezing, hence allowing results to be more specific, allowing a more in depth understanding of the association between income and specific wheezing phenotypes (Subbarao et al., 2009; Savenije et al., 2011; Taylor-Robinson et al., 2015).

4.2. Definitions of wheezing phenotypes

There is no standard method of defining and identifying child wheezing phenotypes. However, data such as age of onset and duration of wheezing or wheezing triggers, such as respiratory infection, are often used to define wheezing phenotypes. Many studies use pre-defined wheezing phenotypes from the literature, often applying similar phenotypes to those first defined by Martinez et al (1995). More recent studies have used data-driven methods such as latent class analysis (LCA) to identify the most common wheezing phenotypes in a given population, this approach is further detailed in Section 4.2.2 (Panico et al., 2014; Deliu et al., 2017).

The following section reviews 1) studies that have used pre-defined wheezing phenotypes based primarily on age of onset and time course and 2) studies that have used LCA to identify wheezing phenotypes based on onset and time course. Table 4.1 provides a summary of these studies.

4.2.1. Pre-defined phenotypes

Since the novel categorisation of wheezing phenotypes adopted by Martinez et al (1995) using the Tuscon study (Table 4.1), there have been further studies that have adopted a similar strategy to classify wheezing.

As an extension to the ISAAC, children were enrolled in the Italian Study of Respiratory Disorders in Childhood and the Environment (SIDRIA) (Rusconi et al., 1999). Two groups of children ages 6 to 7 years and ages 13 to 14 years were recruited in 1994-1995, and parents answered standardised questionnaires including core modules for asthma from the ISAAC (Rusconi et al., 1999). Four mutually exclusive groups were defined in the analysis and these were similar to those used by Martinez: 'no wheezing' (children with no lower respiratory tract

illness with wheezing during the first 2 years, no asthma up to age 6 or 7 and no wheezing in the last 12 months) (82.8%); 'transient early wheezing' (those who had at least one respiratory tract illness with wheezing in the first 2 years of life with no wheezing in the last 12 months) (7.5%); 'persistent wheezing' (those who had at least one respiratory tract illness with wheezing in the first 2 years of life and had wheezing in the last 12 months) (4.1%); 'wheezing of late onset' (those with no lower respiratory tract illness with wheezing in the first 2 years, but who had wheezing in the last 2 years) (5.6%) (Table 4.1)

The Dunedin Multidisciplinary Health and Development Study is a longitudinal, population-based cohort study in New Zealand of children born between April 1972 and March 1973 (Sears et al., 2003). A combination of questionnaires and medical testing was used to assess respiratory function of 613 cohort members. Children were examined every two years between the ages of 3 and 15 years and then at ages 18, 21 and 26 years, for lung function and airway responsiveness tests. Questionnaires that provided detailed information about a child's respiratory health history were completed by parents at ages 9, 11, 13, 15 years with questionnaires being completed by the cohort member themselves at ages 18, 21 and 26. Patterns of wheezing reported by study members or their parents and corresponding frequencies in parentheses were: 'no wheezing' (wheezing not reported at any age) (27.4%); 'persistent from onset' (wheezing present at all ages from onset other than at age nine) (14.5%); 'remission' (the absence of wheezing at a particular age after wheezing was reported at two or more successive prior assessments) (15%); 'relapse' (wheezing reported at two or more successive assessments, then absent at one or more successive assessments, and then reported at all subsequent assessments) (12.4%); 'intermittent' (defined as wheezing that is present at some assessments but not at others, not present at two consecutive assessments and not fitting in previously described patterns) (9.5%); and 'transient wheeze' (wheezing reported at only one assessment over the study period) (21.2%) (Table 4.1).

Kurukulaaratchy et al (2003) used data from a birth cohort that was established on the Isle of Wight in 1989, to prospectively study the natural history of child asthma and its risk factors. Data were collected from 1,536 children born between 1989 and 1990 with follow-up at ages 1, 2, 4 and 10 years. At every follow-up parents answered detailed questionnaires regarding asthma and allergy prevalence for each child with 'current wheeze' defined as wheezing that had occurred in the previous 12 months. At age 10, children were then categorised into groups according to the presence of current wheeze at each follow-up. These groups and their frequency in parentheses were 'non wheezers' (children who never wheezed in the first decade of life) (59.7%), 'early transient wheezers' (wheezing onset during the first 4 years of life that ceased and was not present within 12 months of assessment at age 10) (20.4%), 'persistent wheezers' (children with wheezing onset during the first 4 years who still wheezed at age 10) (12.1%) and 'late onset wheezers' (children with wheezing onset from age 5 onwards who still wheezed at age 10) (7.8%) (Table 4.1).

Lung function and wheezing phenotypes were studied in a population-based birth cohort study, The Manchester Asthma and Allergy Study, by Lowe et al (2005). The study recruited children prenatally in 1995 between 8 to 10 weeks gestation (Lowe et al., 2005). Children's respiratory health was assessed at ages 3 and 5 by use of parent-answered respiratory questionnaires, tests for specific airway resistance, allergic sensitisation tests and airway reactivity tests (Lowe et al., 2005). According to parent-reported history of child wheeze at ages 3 and 5, the following categories were assigned: 'no wheezing' (no wheezing at age 3 and no wheezing ever at age 5) (55.7%), 'transient early wheeze' (no wheeze during the first 3 years and no wheezing in the previous 12 months at age 5) (23.5%); 'persistent wheeze' (wheezing during the first 3 years and wheezing in the previous 12 months at age 5) (15.1%); and, 'late onset wheezing' (no wheeze during the first 3 years and wheezing in the previous 12 months at age 5) (5.8%) (Lowe et al., 2005) (Table 4.1).

Using data from the Canadian National Longitudinal Survey of Children and Youth (NLSCY), Midodzi et al (2008), analysed risk factors associated with wheezing phenotypes. Longitudinal surveys were conducted two years apart at five time points from 1994 to 2003. The sample consisted of 2,711 children who were between the ages of 0 and 23 months at the first survey. Children were classified into mutually exclusive phenotypes, which were defined using the sequence of wheezing outcomes reported in each of the surveys during preschool (0–5 years old) and primary school years (6–9 years old). The phenotypes defined were 'non wheezers' (49.5%); 'preschool wheezers' (children with at least one reported wheezing episode during their preschool years, and no reported wheezing during primary school years) (25.4%); 'primary school wheezers' (children who had not reported any wheezing during preschool years, but reported at least one during their primary school years) (8.5%); 'intermittent wheezers' (children who intermittently reported at least one wheezing episode during both their preschool and primary school years)(6.7%) and 'persistent wheezers' (children with two consecutive reports of wheeze during preschool years and primary school years) (9.9%) (Midodzi et al., 2008) (Table 4.1).

In a recent study using data from the UK based MCS, the dataset used in this thesis, Taylor-Robinson et al (2015) classified wheezing patterns using data from 11,141 children at ages 3, 5 and 7, according to the groups used by Martinez et al (1995). Wheezing was recorded as a dichotomous variable at each age where mothers were asked if their child had wheezing in the last 12 months and then phenotypes classified as follows: 'never wheezed' (66%); 'early remitting' (wheezing at any point up to age 5 but not at age 7) (17.1%); 'transient/intermittent' (intermittent wheezing is defined as wheezing that comes and goes, and transient wheezing as wheezing that was reported at only one assessment point) (4.9%); 'persistent/relapsing' (wheezing present at age 7 to capture persistent and late onset wheezing) (12%) (Taylor-Robinson et al., 2015) (Table 4.1).

4.2.2. Categorisation by Latent Class Analysis (LCA)

LCA is a data-driven technique to explore whether there are groups within the study population with some degree of homogeneity, for example, in terms of wheezing symptoms. When longitudinal data has been collected, LCA uses the responses at each survey for each child, identifying groups that share similar longitudinal response patterns. It relies on the statistical model to generate clusters of phenotypes based on the variables added to the model rather than pre-formulated hypotheses (Brew et al., 2019).

The main assumption of LCA is that the study population is composed of distinct subsets that are referred to as 'latent classes' based on the distribution of some key variables present in these subsets (Panico et al., 2014). LCA estimates two sets of parameters for each class: the conditional response probability, which gives the probability of observing a specific response pattern; and the latent class prevalence, which gives the proportion of children in that subset (Panico et al., 2014). To identify the correct number of latent classes in the study population, models are fitted repeatedly, increasing the number of classes in a stepwise approach (Panico et al., 2014; Brew et al., 2019). The latent class model aims to determine the minimum number of latent classes that best describe the observed patterns of responses (to questions about wheeze) in the data (Panico et al., 2014; Brew et al., 2019).

Spycher et al (2008) used LCA to identify distinct classes of wheeze underlying the observed heterogeneity in data from 1,650 children between the ages of 0 and 5 recruited into a population based cohort study in Leicestershire, United Kingdom in 1990 (Spycher et al., 2008). Parents completed postal questionnaires on respiratory symptoms, exposures and sociodemographic factors in 1990, 1998 and 2003, with a nested sample of the children being invited for physiological testing and interviews between 1992 and 1994. Only data from the 1990 survey and the 1992/1994 nested sample were used for phenotype classification. Because

LCA was applied to both respiratory symptoms (wheezing, cough, shortness of breath) and physiological measurements (allergic sensitisation, bronchial responsiveness, forced expiration volume), the resulting phenotypes were comprised of both of these elements and were (with prevalence in parentheses): 'persistent cough' (cough apart from colds in both surveys, and wheezing was commonly reported) (30.4%); 'transient cough' (cough apart from colds in the first survey, and wheezing was rarely reported) (27.5%); 'atopic persistent wheeze' (wheezing was commonly reported in both surveys, and sensitisation to at least one allergen was likely) (18.2%); 'non-atopic persistent wheeze' (attacks of wheezing were likely at both surveys, and atopic sensitisation was rare) (14.7%); and 'transient viral wheeze' (attacks of wheeze tended to be reported before the first survey, and if reported at the first survey these attacks were infrequent and had subsided by the second survey) (11.0%) (Table 4.1). Although the phenotypes classified in this study are more extensive than the Martinez (1995) classification and cannot be directly compared as they include additional physiological factors such as atopy and viral coughs, the distinction made between persistent and transient phenotypes remains.

Using data from the ALSPAC study, a UK-based cohort study that recruited 14,541 pregnant women during 1991 and 1992, Henderson et al (2008) used LCA to identify wheezing phenotypes. Mothers were sent self-completion questionnaires about the health of their children at ages 6, 18, 30, 42, 54, 69, 81 and 91 months. Data collected included data on wheezing in the last 12 months at approximately 12 month intervals. Six phenotypes were identified with prevalence in parentheses: 'Never or infrequent wheeze' (approximately 10% prevalence of wheezing at 6 months with declining prevalence of sporadic wheeze thereafter and included subjects who never reported wheeze at all (59.3%); 'Transient early wheeze' (had 50–60% prevalence up to 18 months, declining to low prevalence from 42 months (16.3%); 'Prolonged early wheeze' (had a peak prevalence of around 65% at 30 months, declining to low prevalence from 69 months onwards) (8.9%); 'Intermediate-onset wheeze' (had a low

prevalence up to 18 months, rising rapidly to high prevalence from age 42 months onwards) (2.7%); 'Late-onset wheeze' (had approximately 20% prevalence up to 42 months, rising to 50% or higher prevalence thereafter) (6.0%); and 'Persistent wheeze' (had 65% prevalence at 6 months with approximately 90% prevalence thereafter) (6.9%) (Henderson et al., 2008) (Table 4.1).

In another longitudinal LCA, Savenije et al (2011) did a comparison study, comparing data from the PIAMA study, a multicentre birth cohort study that selected 4,146 pregnant women in the Netherlands between 1996 and 1997, to data from the ALSPAC study described previously in Henderson et al (2008). In the PIAMA study, parents completed questionnaires about the health of their children at 3, 12, 24, 36, 48, 60, 72, 84 and 96 months after birth, with clinical examinations taking place at ages 4 and 8 years. Wheezing was defined to be present if the following two questions were answered with a yes: "*Has your child had wheezing or whistling in the chest at any time in the past*?" and "*Has your child had wheezing or whistling in the chest in the past 12 months*?". Five phenotypes were derived from the PIAMA study, with identical phenotypes as those identified in the ALSPAC data, with the exception of 'prolonged early wheeze' which is not in the PIAMA model. These phenotypes were: 'Never or infrequent wheeze' (75%), 'Transient early wheeze' (16.7%), 'Intermediate-onset wheeze' (3.1%), 'Late-onset wheeze' (1.7%) and 'Persistent wheeze' (3.5%) (Savenije et al., 2011) (Table 4.1).

Lodge et al (2014) explored wheezing phenotypes using the Melbourne Atopy Cohort Study which is a high-allergy-risk birth cohort that collected data on Australian children over the first 7 years with additional follow-up at age 12. The data used comprised of 640 children born to pregnant mothers recruited between 1990 and 1994. Data were collected every 4 weeks up to age 15 months, at 18 months, then yearly from ages 2 to 7 years, and then once at age 12 years. Mothers reported the frequency of wheezing in days (up to age 15 months of age), the frequency of wheezing in episodes and a yes or no response to whether their child had wheezing in the

previous 12 months (in the ages 3 to 7 years follow-up). The LCA identified the following phenotypes: 'never or infrequent wheeze' (42.7%), 'early transient wheeze' (appeared in the first 12 months and resolved at around 3 years) (27.5%), 'early persistent wheeze' (appeared in the first 6 months) (5.6%), 'intermediate onset wheeze' (emerged around 18 months) (20.7%), and 'late onset wheeze' (occurred after age 4) (3.5%) (Lodge et al., 2004) (Table 4.1).

Panico et al (2014) aimed to identify longitudinal trajectories of wheeze in early childhood using data from the MCS, the same dataset that was used in Taylor-Robinson (2015), however the latter used predefined phenotypes. LCA was applied to data on wheezing at ages 3, 5 and 7 years including severity and parental reports of atopic symptoms. The phenotypes identified were (with prevalence in parentheses): low levels of both wheeze and other atopic symptoms (characterised by low levels of all symptoms) (54%); low levels of wheeze and high levels of other atopic symptoms (characterised by low levels of wheeze at all ages, however this group is also characterised by high levels of eczema and/or hay fever, with about 85% of the sample reporting eczema or hay fever by age 7) (29%); high levels of both wheeze and other atopic symptoms (the highest levels of wheeze and the highest proportion of children reporting eczema and/or hay fever. Children in this group report the most severe wheezing symptoms and the most limitations due to wheezing) (9%); and lastly high levels of wheeze and low levels of other atopic symptoms (this group had relatively high levels of wheeze, although both overall levels and severity of symptoms are below that of group described just before) (8%) (Table 4.1) (Panico et al., 2014). These classifications are different to the other studies reviewed so far as they are based on severity of wheezing rather than time of onset and therefore cannot be directly compared.

While the studies reviewed above have used or identified different wheezing phenotypes with a variety of data and definitions, through LCA or predefined categories of wheezing, common phenotypes emerge:

- never wheezed
- early remitting
- persistent wheeze
- late onset
- transient/intermitting

Table 4.1: Wheezing phenotypes defined by age of onset using clinical definitions and derived from Latent Class Analysis

Cohort/Study	Population	Sample size (N)	Age/ Year of follow-up	Method	Phenotypes described
Tuscon Children's Respiratory Study (TCRS) (Martinez et al 1995)	Children born 1980-1984 Tuscon, Arizona, USA	826	3 and 6 years	Age based	Never wheeze (51%) Early transient 20%) Persistent (14%) Late onset (15%)
Italian Study of Respiratory Disorders in Childhood and the Environment (SIDRIA) (Rusconi et al 1999)	Children 6-7 years, born 1994-1995, Italy	16,333	N/A	Age based	Never wheeze (83%) Transient early (7%) Persistent (4%) Late onset (6%)
Dunedin Multidisciplinary Study (Sears et al 2003)	Children born 1972 -1973, New Zealand	631	9, 11, 13, 15,18, 21 and 26 years	Age based	Never wheeze (27.4%) Transient (21.2%) Persistent (14.5%) Relapse (12.4%) Remission (15%) Intermittent (9.5%)
Isle of Wight birth cohort (Kurukulaaratchy et al 2003)	Children born 1989-1990, Isle of Wight	1,536	1, 2, 4 and 10 years	Age based	Never wheeze (59.7%) Early transient wheeze (20.4%) Persistent wheeze (12.1%) Late onset wheeze (7.8%)
The Manchester Asthma and Allergy Study (Lowe et al 2004)	Children recruited in-utero between 8-10 weeks gestation, UK	665	3 and 5 years	Age based	No wheezing (55.7%) Transient early wheeze (23.5%) Persistent wheeze (15.1%) Late onset wheeze (5.8%)
Canadian National Longitudinal Survey of Children and	Children 0-23 months in 1994-1995, Canada	2,711	Five cycles of surveys conducted in 1994/1995, 1996/1997, 1998/1999,	Age Based	Non wheezers (49.5%) Preschool wheezers (25.4%),

Cohort/Study	Population	Sample size (N)	Age/ Year of follow-up	Method	Phenotypes described
Youth (NLSCY) (Midodzi et al 2008)			2000/2001 and 2002/2003		Primary school wheezers (8.5%) Persistent wheezers (9.9%)
Millennium Cohort Study (MCS) (Taylor-Robinson et al 2015)	Children born 2000-2001, UK	11,141	9 months, 3, 5 and 7 years	Age based	Never wheeze (66%) Early remitting wheeze (17.1%) Transient/ Intermittent wheeze (4.9%) Persistent (12%)
Spycher et al 2008	Children 0-5 years recruited in 1990, UK	319	1990, 1998 and 2003	LCA	Persistent cough (30.4%) Transient cough (27.5%) Transient viral wheeze (11.0%) Atopic persistent wheeze (18.2%) Non-atopic persistent wheeze (14.7%)
British Avon Longitudinal Study of Parents and Children (ALSPAC) (Henderson et al 2008)	Children whose mothers were pregnant during 1991 and 1992, UK	6,625	6, 18, 30, 42, 54, 69, 81 and 91 months	LCA using age-specific data	Never wheeze (59%) Transient early wheeze (16%) Prolonged early wheeze (9%) Intermediate-onset wheeze (3%) Persistent wheeze (7%) Late-onset wheezing (6%)
Netherlands Prevention and Incidence of Asthma and Mite Allergy (PIAMA) Study (Savenije et al 2011)	Children whose mothers were pregnant between 1996 and 1997, Netherlands	2,810	3 months, 1, 2, 3, 4, 5, 6, 7, and 8 years	LCA using age-specific data	Never wheeze (75%) Transient early wheeze (17%) Intermediate-onset wheeze (3%) Persistent wheeze (4%) Late-onset wheeze (2%)
Melbourne Atopy Cohort Study (Lodge et al 2014)	Children born to pregnant mothers recruited between 1990 and 1994, Australia	640	Every 4 weeks up to 15 months, at 18 months, 3, 4, 5, 6, 7 and 12 years	LCA using age-specific data	Never wheeze (42.7%) Early transient wheeze (27.5%) Early persistent wheeze (5.6%) Intermediate onset wheeze (20.7%) Late onset wheeze (3.5%)
Millennium Cohort Study	Children born between 2000 and 2001, UK	11,632	Data from age 3, 5 and 7	LCA using age-	Low level wheeze and other atopic symptoms (84%)

Cohort/Study	Population	Sample size (N)	Age/ Year of follow-up	Method	Phenotypes described
(MCS) (Panico et al 2014)				specific data	Low level wheeze and high level atopic symptoms (29%) High levels wheeze and high levels atopic symptoms (9%) High levels wheeze and low levels atopic symptoms (8%)

4.2.3. The association between socioeconomic status and wheezing phenotype

This section outlines previous research that has explored the association between socioeconomic status and wheezing phenotypes. Although the main focus is the role income itself, there are very few studies that have studied the association between income and wheezing phenotype. In Chapter 3, there was extensive previous literature which had explored the association between low income and adverse asthma-related outcomes, but analysis only included dichotomous measures of asthma-related outcomes.

A previous analysis of the MCS by Panico et al (2014) identified wheezing phenotypes based on the severity (high versus low levels) of atopy and wheezing as reported at age 3, 5 and 7 as described in Section 4.2.2. This study provided evidence of how socioeconomic characteristics (measured by parental income and educational level) and early markers of well-being, such as birth weight, were associated with these phenotypes. Socioeconomic measures included income which was modelled as the log of annual parental income and highest level of educational qualification for resident parents when the child was age 9 months (Panico et al., 2014). Results showed that children with low levels of wheeze but high levels of other atopic symptoms tended

to belong to the most socioeconomically advantaged group, while children with high levels of wheeze and low levels of other atopic symptoms belonged to the least advantaged group (Panico et al., 2014).

Taylor-Robinson et al (2015) study is another MCS study which analysed the association between socioeconomic status and wheezing phenotype, as described in Section 4.2.1) (Table 4.1). In this study, mother's education was used as the measure of socioeconomic status. Key findings were that children of mothers with no and low educational qualifications are 40% more likely to have early transient wheeze and 30% more likely to have persistent wheeze compared to children of mothers educated to degree level, after adjusting for many other covariates (Taylor-Robinson et al., 2015). Galobardes et al (2015) investigated the association between paternal education and wheezing phenotypes using the ALSPAC study and phenotypes derived by Henderson et al (2008). They found that children whose fathers had a low level of education (Ordinary level and below) were more likely to have persistent wheezing (OR: 1.46; 95% CI 1.12;1.92) compared to highly educated fathers.

These three studies suggest that wheezing phenotypes are at least in part determined by level of socioeconomic status and in particular, that lower income has been found to be associated with more frequent and more persistent wheezing. However, these studies used data up to age 7 and would benefit from a longer follow-up period to assess patterns of wheezing more accurately. In addition, Panico et al (2014) that used income as a measure of socioeconomic status used income at baseline (at 9 months), which may not be an accurate marker of socioeconomic income compared to permanent income. Furthermore, Galobardes et al (2015) and Taylor-Robinson et al (2015) did not include income as a determinant in their analysis. Therefore this chapter adds to the literature by using a longer follow-up period in classifying wheezing phenotypes, up to age 11, and explores the impact of permanent income on wheezing phenotypes.

4.3. Methods

4.3.1. Theoretical model

Asthma-related outcomes are estimated using the health capital model developed by Grossman (1972a, 1972b and 2000) (Chapter 2, Section 2.11.1) expressed by the following equation:

$$H_{it}^{c} = \alpha + \beta lnY_{it} + \gamma X_{it}^{p} + \theta X_{it}^{C} + \varepsilon_{it} \quad (4.1)$$

$H_{it}^{c}$is child i's distinct wheezing phenotype, Y_{it} is income; X_{it}^{p} represents a set of variables composed of parental characteristics other than income, and X_{it}^{C} is a set of child characteristics.

4.3.2. Model specification and estimation strategy

4.3.2.1. Defining wheezing phenotypes for analysis

Wheezing in the last 12 months (measures at ages 3, 5, 7 and 11) was selected from the MCS to be the most appropriate measure for wheezing in children for this analysis. As the MCS surveys are timed with at least two years between surveys, responses to the questions on wheezing in the past year did not refer to overlapping time periods making it possible to define mutually exclusive wheezing phenotypes. As was demonstrated in the literature review, there is a lack of uniformity in defining phenotypes associated with childhood wheezing but there exists some commonly used groups. This is in part due to differences in data i.e. the number of surveys completed during follow-up, the age at which surveys were conducted, and the spacing between surveys.

Therefore, for the purposes of this analysis, wheezing phenotypes are defined as in Table 4.2 for the base case, similar to the categories used by Martinez et al (1995). Applying these phenotypes to the MCS data using the dichotomous coding detailed in Appendix C-1,

frequencies of each wheezing phenotype were calculated and displayed in the last column of Table 4.2.

Table 4.2: Wheezing phenotypes defined in the base case analysis

Phenotype	Description	Prevalence of wheezing
Never wheezed	No report of wheezing at any age	6,794 (66.2%)
Early remitting wheeze	Includes children who had wheezing at age 3 but not at age 5, 7 and 11	882 (8.6%)
Late onset wheeze	Includes children who reported wheezing for the first time at age 5, 7 or 11, but not before	1,099 (10.7%)
Persistent wheeze	Children who had wheezing at age 3 and at age 5, and/or 7 and/or 11	1,482 (14.4%)

It can be seen that the most common phenotype using MCS data is 'Never wheezed', the other three phenotypes have similar prevalence with 'Early remitting' wheeze accounting for 9% of the population, 'Persistent' wheeze accounting for 14% of the population and 'Late onset' wheeze accounting for 11% of the population (Table 4.2, Figure 4.2).

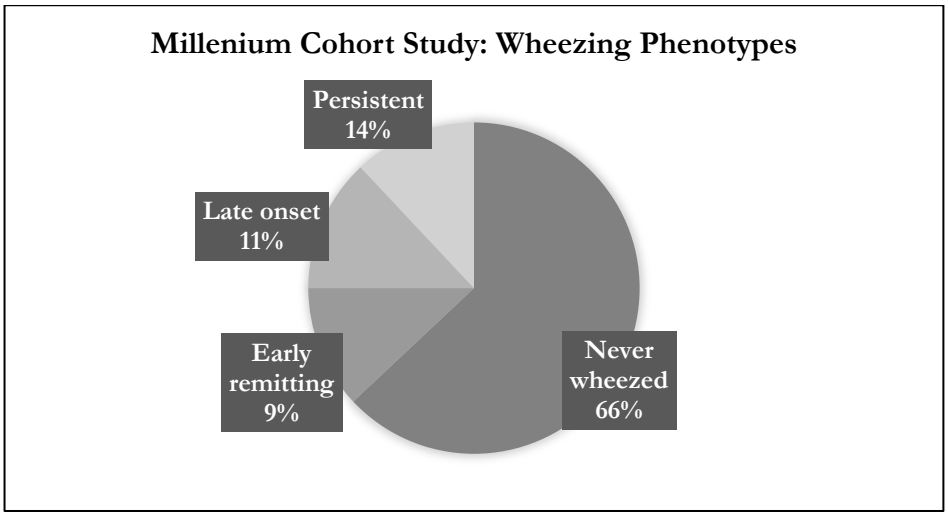


Figure 4.1: MCS prevalence of wheezing phenotype

4.3.2.2. Income

The main exposure in the base case analysis is family income, which is operationalised as permanent income. To recap, permanent income is calculated as average annual household income up to and including income at the age at which the wheezing outcome was measured, expressed in 2011 prices and in its natural logarithmic form (Chapter 2, Section 2.9.1).

4.3.2.3. Model specification and estimation strategy

Multinomial logistic regression analysis was carried out where the dependent variable was wheezing phenotype with each child belonging to one of the groups: never wheezed, early remitting wheeze, late onset wheeze or persistent wheeze. Never wheezed was set as the reference case in the models. The modelling approach used in the cross-sectional analysis (Chapter 3, Section 3.2.3) has been applied here i.e. a five-stage stepwise regression analysis. At each step, the set of variables associated with each model specification, as detailed in Table 4.3, was added to the model. A priori variables were included in each model specification as they are background factors that are seen as unmodifiable. Explanatory variables in specifications 3, 4 and 5 were only included if they were significantly associated with wheezing phenotype (significance was defined by a coefficient having a Wald test p-value <0.1), otherwise variables were removed from the models. Both multinomial coefficients and relative risk ratios (RRRs) were estimated for all models.

Table 4.3: Model specification

Model specification	Description of variables included in model
Specification 1	Unadjusted association between permanent income and wheezing phenotype
Specification 2	Specification 1+ a priori variables: Ethnicity, sex, child age, birthweight, family history of asthma Number of siblings at 9 months
Specification 3	Specification 2 + other socioeconomic variables: mother's highest level of education at 9 months, mother's age at time of birth, housing tenure at 9 months

Specification 4	Specification 3 + mother and child health related variables: smoking during pregnancy, alcohol consumption during pregnancy, mother's BMI at delivery, delivery mode, breast feeding duration, mother's general health at age 3, mother's mental health at age 3
Specification 5	Specification 4 + environmental factors: damp or condensation in the home at 9 months, exposure to cigarette smoke at 9 months , exposure to fumes from heating at 9 months , rural-urban morphology at 9 months

4.3.3. Sensitivity analysis

In the base case definition of wheezing phenotypes, early remitting wheeze phenotype was defined as children who had wheezing in the previous 12 months at age 3, but not at age 5 and over. In the first sensitivity analysis early remitting wheeze included children who had wheezing at age 3 or 5, but not age 7 or 11. Because this cut-off is at a later age, it in turn affects the definition of late onset wheezing. Therefore, late onset wheeze in this sensitivity analysis includes children who had wheezing for the first time at age 7 or 11 and not before.

As a second sensitivity analysis, income at age 9 months was used as an alternative to permanent income. I use income at 9 months as a measure of baseline income, to investigate the extent to which income in the very early years may affect wheezing throughout childhood. This is also consistent with the Panico et al (2014) which included family income at 9 months in their analysis of wheezing phenotypes and allows for comparison of results to some extent.

As a third sensitivity analysis, I differentiate between children with a family history of asthma i.e. either mother or father reporting asthma and those without a family history of asthma. This sensitivity analysis was aimed at investigating whether family history of asthma had an impact on the association between family income and wheezing phenotype. This analysis will aim to capture any hereditary associations with phenotype, however it is a very simplified measure.

4.4. Results

4.4.1. Descriptive analysis

Table 4.4 shows the prevalence of wheezing phenotypes across the income distribution where income is divided into permanent income quintiles. The proportion of children with no wheeze increases as family income increases. Similarly, the proportion of children with wheezing of any of the other phenotypes decreases as family income increases, with prevalence across income quintiles ranging from 11.0% to 13.2% from the lowest to the highest income quintile for early remitting wheeze, 11.5% to 14.7% for late onset wheeze, and from 9.6% to 13.6% for persistent wheeze (Table 4.4).

Table 4.4: Wheezing phenotype by income quintile

Income quintile	No wheezing	Early remitting	Late onset	Persistent
Lowest quintile	58.5% (1,089)	13.2% (218)	14.7% (274)	13.6% (282)
Second quintile	59.6% (1,166)	12.4% (261)	14.3% (266)	14.2% (262)
Third quintile	64.3% (1,271)	11.9% (237)	12.5% (260)	11.3% (224)
Fourth quintile	66.6% (1,355)	11.1% (225)	12.4% (266)	9.9% (213)
Highest quintile	67.9% (1,379)	11.0% (225)	11.5% (242)	9.6% (205)

4.4.2. The association between income and wheezing phenotype

Relative risk ratios (RRRs) and marginal effects of permanent income on wheezing phenotype (for the base case) are shown in Table 4.5. For each stage of the estimation strategy (1-5), marginal effects for each phenotype are reported above the RRRs, with their 95% confidence interval reported in parentheses. Never wheezed is the reference category.

Table 4.5: The association between income and wheezing phenotype (base case): Marginal effects and RRRs

Model specification	Never wheezed	Early Remitting	Late onset	Persistent
Specification 1	reference	-0.018** 0.76 (0.64-0.90)	-0.021** 0.76 (0.65-0.88)	-0.035*** 0.66 (0.56-0.77)
Specification 2	reference	-0.028*** 0.68 (0.57-0.82)	-0.020** 0.75 (0.63-0.89)	-0.035*** 0.64 (0.54-0.76)
Specification 3	reference	0.002 0.91 (0.69-1.20)	-0.028* 0.72 (0.56-0.94)	-0.037*** 0.64 (0.49-0.84)
Specification 4	reference	0.016 1.11 (0.81-1.50)	-0.027* 0.76 (0.57-1.03)	-0.016 0.82 (0.60-1.11)
Specification 5	reference	0.014 1.07 (0.79-1.45)	-0.028* 0.77 (0.57-1.03)	-0.015 0.83 (0.61-1.12)

*p-value<0.1; **p-value<0.05; ***p-value<0.01; marginal effects reported in top row;
RRRs reported in lower row with 95% CI in parentheses.

In the unadjusted model (specification 1), the marginal effect of early remitting wheeze, -0.018, can be interpreted as a 1% increase in income is associated with a 0.018 decrease in the probability of a child having early remitting wheeze, compared to never wheezing. This effect is statistically significant (p-value:0.04)[25]. On adding a priori variables (specification 2) the negative marginal effect associated with early remitting phenotype increased in absolute value to 0.028 (p-value<0.001). In specification 3, with the addition of other socioeconomic measures, the association between income and early remitting wheeze became positive although the effect was small and not statistically significant (marginal effect: 0.002; p-value: 0.36), this did not change in specification 4 and 5.

For late onset wheeze, the marginal effect of income was strong and statistically significant (marginal effect: -0.021; p-value:0.03). The magnitude of the marginal effect remained similar

[25] In terms of RRR, a 1% increase in income results in the relative risk of a child having early remitting wheeze to be 0.76 (95% CI: 0.64,0.90) times more likely compared to having no wheeze. This interpretation can be applied for all other RRRs.

across model specifications, however in specification 4 and 5, the statistical significance was weaker.

For persistent wheeze, the magnitude of the marginal effect was larger than for early remitting and late onset wheeze and was highly statistically significant (p-value<0.001) for specification 1, 2 and 3 (Table 4.5). In specification 4 and 5 the marginal effect associated with persistent wheeze more than halved in magnitude to -0.016 (p-value: 0.30)and was not statistically significant. Overall it can be seen in the fully adjusted models, that there is evidence of an association between low family income and late onset wheeze in children, although the evidence is weak. There is no strong evidence to support the association between family income and early remitting wheeze and persistent wheeze.

4.4.3. The association between other factors and wheezing phenotype

Factors other than income that were associated with early remitting wheeze were ethnicity, with a higher marginal probability in black Caribbean children compared with white children (marginal effect: 0.10; p-value: 0.01; 95% CI:1.38,4.18)[26]. A family history of asthma was also positively associated with early remitting wheeze (marginal effect: 0.02; 95% CI:1.30,1.83, p-value:0.01). Factors that decreased the probability of early remitting wheeze were having one sibling compared with no siblings (marginal effect:-0.02; 95% CI:1.01,1.48; p-value:0.03), breastfeeding for 4-6 months (marginal effect:-0.04; 95% CI:0.48,0.94; p-value:0.02) or more than 6 months (marginal effect:-0.03; 95% CI:0.54,0.93; p-value:0.04) compared with never

[26] See Appendix C-2 for full results.

breastfeeding, and living in a rural setting compared to urban setting (marginal effect: -0.027; 95% CI:0.55,1.02; p-value: 0.05).

Factors positively associated with late-onset wheeze were being male (marginal effect: 0.03; 95% CI:1.19,1.63; p-value:0.003) and family history of asthma (marginal effect: 0.02; 95% CI:1.28,1.79; p-value:0.02). Having one sibling (marginal effect:-0.02; 95% CI:0.70,1.02; p-value:0.02), two (marginal effect:-0.01; 95% CI:0.67,1.15; p-value:0.05) or three (marginal effect:-0.04; 95% CI:0.45,1.01; p-value:0.03;) siblings compared with no siblings decreased the probability of late-onset wheezing.

For persistent wheeze, being male (marginal effect: 0.06; p-value:0.001; 95% CI: 1.70,2.34), low birth weight (marginal effect: 0.05; 95% CI: 1.12,2.28; p-value:0.02), family history of asthma (marginal effect: 0.09; 95% CI:2.14,3.01; p-value:0.001), planned c-section (marginal effect: 0.04; 95% CI:1.10,1.88; p-value:0.01) and emergency c-section (marginal effect: 0.05; 95% CI:1.27,2.01; p-value:0.001), mothers with medium (marginal effect: 0.03; 95% CI:1.19,1.71; p-value:0.001) and high distress levels (marginal effect:0.07; 95% CI:0.93,1.49; p-value:0.03), fume exposure (marginal effect: 0.02; 95% CI:1.09,1.50; p-value:0.01) were all positive risk factors. Living in a mixed rural-urban setting (marginal effect:-0.03; 95% CI:0.52,0.98; p-value:0.02;) rather than an urban area decreased the probability of a child having persistent wheeze.

4.4.4. Sensitivity analysis

In the first sensitivity analysis, phenotypes were defined using different cut-offs (Table 4.6). The cut-off for the early remitting phenotype was age 7 (i.e. wheezing in the last 12 months at age 3 or age 5 but not at 7 and 11); late onset wheeze was defined as wheezing reported for the first time at age 7 or 11 and not before).

In specification 1, the unadjusted model, a 1% increase in income was associated with a significant decrease in the probability of a child having early remitting wheeze of 0.05 (marginal effect: -0.05; 95% CI:0.61, 0.80; p-value<0.001). From specification 3 to 5, the marginal effect was no longer statistically significant and this pattern is broadly similar to the base case analysis where coefficients lose their statistical significance in specification 3 onwards (Table 4.7).

Table 4.6: Wheezing phenotype defined in sensitivity analysis

Phenotype	Description	Prevalence of wheezing
Never wheezed	No report of wheezing at any age	6,794 (66.2%)
Early remitting	Includes children who had wheezing at age 3 and 5 but not at 7 and 11	1,616 (15.8%)
Late onset wheezing	Includes children who reported wheezing for the first time at age 7 or 11 but not before	687 (6.7%)
Persistent wheezing	Children who had wheezing at age 3 and/or 5 and then at age 7 and/or 11	1,160 (11.3%)

Likewise, the association between income and late onset wheezing was not statistically significant in the unadjusted model. The marginal effect was very small and remains small and not statistically significant in specification 5 (marginal effect: -0004; 95% CI: 0.61, 1.17; p-value: 0.67). This was in contrast to the base case analysis where the association between income and late-onset wheeze was statistically significant, although the association was weak. The association between income and persistent wheezing was statistically significant in the unadjusted model (marginal effect:-0.03; CI:0.60, 0.83; p-value:0.001). This association was fairly constant and remains statistically significant until specification 5 (marginal effect:-0.02; CI:0.5, 0.81; p-value:0.001) in contrast to the base case results which showed no association in the fully adjusted model. Full results that show the association of other risk factors and wheezing phenotype are reported in Appendix C-2.

Table 4.7: The association between income and wheezing phenotype (cut-off age 7 for early remit): marginal effects and RRRs

Model specification	No wheeze	Early Remitting	Late onset	Persistent
Specification 1	reference	-0.05*** 0.69 (0.61-0.80)	-0.001 0.87 (0.73-1.05)	-0.03*** 0.71 (0.60-0.83)
Base case		-0.018** 0.76 (0.64-0.90)	-0.021** 0.76 (0.65-0.88)	-0.035*** 0.66 (0.56-0.77)
Specification 2	reference	-0.038*** 0.64 (0.55-0.75)	-00002 0.87 (0.70-1.07)	-0.024*** 0.69 (0.59-0.82)
Base case		-0.028*** 0.68 (0.57-0.82)	-0.020** 0.75 (0.63-0.89)	-0.035*** 0.64 (0.54-0.76)
Specification 3	reference	-0.019 0.81 (0.64-1.02)	-0.007 0.80 (0.58-1.10)	-0.023*** 0.64 (0.49-0.82)
Base case		0.002 0.91 (0.69-1.20)	-0.028* 0.72 (0.56-0.94)	-0.037*** 0.64 (0.49-0.84)
Specification 4	reference	-0.003 0.94 (0.73-1.20)	-0.002 0.98 (0.71-1.36)	-0.023** 0.82 (0.63-1.07)
Base case		0.016 1.11 (0.81-1.50)	-0.027* 0.76 (0.57-1.03)	-0.016 0.82 (0.60-1.11)
Specification 5	reference	-0.017 0.83(0.67-1.02)	-0.004 0.84 (0.61-1.17)	-0.020*** 0.64 (0.50-0.81)
Base case		0.014 1.07 (0.79-1.45)	-0.028* 0.77 (0.57-1.03)	-0.015 0.83 (0.61-1.12)

**p-value<0.05; ***p-value<0.01; marginal effects reported in top row;
RRRs reported in bottom row with 95% CIs in parentheses.

Table 4.8 reports the sensitivity analysis results where family income was measured at age 9 months compared to permanent income as in the base case analysis. In specification 1 and 2 the marginal effect of income on early remitting wheeze was negative and statistically significant (marginal effect:0.025; 95% CI:0.65,0.82; p-value<0.001). The association was no longer significant for specifications 3-5, similar to the base case results. The association between income at age 9 months and late onset wheeze was negative and statistically significant in specification 1 and 2 (marginal effect:0.01; 95% CI:0.72,0.92; p-value:0.038). After adding additional explanatory variables in specification 3-5, the effect disappeared completely whereas a weak association remained in the base case analysis. The association between income at age 9 months and persistent wheeze was statistically significant in the unadjusted model which remained when a priori variables and other determinant variables were added in specifications

2 and 3 (marginal effect:-0.02; 95 CI: 0.70,0.94; p-value:0.03), comparable to the base case except the magnitude in the latter was larger. The effect disappeared in specification 4 and 5.

Table 4.8: The association between income (at 9 months) and wheezing phenotype: Marginal effects and RRRs

Model specification	No wheeze	Early Remitting	Late onset	Persistent
Specification 1	reference	-0.020*** 0.77 (0.69-0.87)	-0.015** 0.82 (0.73-0.93)	-0.021*** 0.77 (0.69-0.86)
Base case		-0.018** 0.76 (0.64-0.90	-0.021** 0.76 (0.65-0.88)	-0.035*** 0.66 (0.56-0.77)
Specification 2	reference	-0.025*** 0.73 (0.65-0.82)	-0.014** 0.81 (0.72-0.92)	-0.020*** 0.76 (0.67-0.85)
Base case		-0.028*** 0.68 (0.57-0.82)	-0.020** 0.75 (0.63-0.89)	-0.035*** 0.64 (0.54-0.76)
Specification 3	reference	-0.013 0.84 (0.70-0.98)	-0.016 0.82 (0.71-0.95)	-0.015** 0.81 (0.70-0.94)
Base case		0.002 0.91 (0.69-1.20)	-0.028* 0.72 (0.56-0.94)	-0.037*** 0.64 (0.49-0.84)
Specification 4	reference	-0.010 0.86 (0.72-1.03)	-0.017 0.82 (0.69-0.98)	-0.009 0.86 (0.72-1.03)
Base case		0.016 1.11 (0.81-1.50)	-0.027* 0.76 (0.57-1.03)	-0.016 0.82 (0.60-1.11)
Specification 5	reference	-0.010 0.87 (0.73-1.02)	-0.014 0.85 (0.72-0.99)	-0.013 0.84 (0.72-0.97)
Base Case		0.014 1.07 (0.79-1.45)	-0.028* 0.77 (0.57-1.03)	-0.015 0.83 (0.61-1.12)

**p-value<0.05; ***p-value<0.01; marginal effects reported in top row;
RRRs reported in bottom row with 95% CIs in parentheses.

For the third sensitivity analysis, cohort children were grouped according to whether or not they had a family history of asthma, and the multinomial models were fitted in these groups separately.

- **No family history of asthma**

In this subgroup (n=7,362), there was a no statistically significant association between income and early remitting wheeze in the unadjusted model (specification 1) nor in the fully adjusted model (specification 5) (Table 4.9). For persistent wheezing, there was a strong association between income and persistent wheeze which disappears after specification 3 in a similar way

to the results for the base case (Table 4.9). The association between income and late onset wheezing was constant from specification 1 to 5 with a weak association like the base case results, except the weak association disappears altogether in specification 5 (Table 4.9).

- **Family history of asthma**

In this subgroup (n=2,559), the association between income and early remitting wheeze was not statistically significant (with the exception of specification 2). For persistent wheeze, the association disappears after specification 3 as with the base case and subgroup of no family history of asthma although the coefficients are much larger. In all specifications, there was no association between income and late onset wheeze (Table 4.10).

Table 4.9: The association between income and wheezing phenotype (no family history of asthma sub-group): Marginal effects and RRRs

Model specification	No wheeze	Early Remitting	Late onset	Persistent	Total
Sample size (n)	4,928	822	909	703	7,362
Specification 1	reference	-0.014 0.80 (0.66-0.95)	-0.020** 0.76 (0.64-0.92)	-0.039*** 0.59 (0.50-0.71)	
Base case		-0.018** 0.76 (0.64-0.90)	-0.021** 0.76 (0.65-0.88)	-0.035*** 0.66 (0.56-0.77)	
Specification 2	reference	-0.021** 0.73 (0.60-0.89)	-0.020 0.75 (0.61-0.92)	-0.042*** 0.56 (045-0.68)	
Base case		-0.028*** 0.68 (0.57-0.82)	-0.020** 0.75 (0.63-0.89)	-0.035*** 0.64 (0.54-0.76)	
Specification 3	reference	0.003 0.94 (0.69-1.29)	-0.024 0.75 (0.56-1.02)	-0.033** 0.64 (0.45-0.91)	
Base case		0.002 0.91 (0.69-1.20)	-0.028* 0.72 (0.56-0.94)	-0.037*** 0.64 (0.49-0.84)	
Specification 4	reference	0.016 1.10 (0.78-1.55)	-0.037** 0.70 (0.49-0.99)	-0.010 0.85 (0.56-1.29)	
Base case		0.016 1.11 (0.81-1.50)	-0.027* 0.76 (0.57-1.03)	-0.016 0.82 (0.60-1.11)	
Specification 5	reference	0.010 1.04 (0.76-1.41)	-0.020 0.80 (0.59-1.10)	-0.026 0.71 (0.49-1.02)	
Base case		0.014 1.07 (0.79-1.45)	-0.028* 0.77 (0.57-1.03)	-0.015 0.83 (0.61-1.12)	

**p-value<0.05; ***p-value<0.01; marginal effects reported in top row;
RRRs reported in bottom row with 95% CIs in parentheses.

Table 4.10: The association between income and wheezing phenotype (with family history of asthma sub-group): Marginal effects and RRRs

Model specification	No wheeze	Early Remitting	Late onset	Persistent	Total
Sample size (n)	1,333	344	399	483	2,559
		-0.032	-0.024	-0.023	
Specification 1	reference	0.68 (0.50-0.92)	0.73 (0.56-0.96)	0.76 (0.58-0.99)	
		-0.018**	-0.021**	-0.035***	
Base case		0.76 (0.64-0.90)	0.76 (0.65-0.88)	0.66 (0.56-0.77)	
		-0.048***	-0.021	-0.017	
Specification 2	reference	0.59 (0.42-0.82)	0.73 (0.54-0.99)	0.77 (0.57-1.02)	
				-0.035***	
		-0.028***	-0.020**	0.64 (0.54-0.76)	
Base case		0.68 (0.57-0.82)	0.75 (0.63-0.89)		
		0.008	-0.043	-0.072**	
Specification 3	reference	0.87 (0.56-1.36)	0.61 (0.40-0.92)	0.54 (0.35-0.82)	
		0.002	-0.028*	-0.037***	
Base case		0.91 (0.69-1.20)	0.72 (0.56-0.94)	0.64 (0.49-0.84)	
		0.015	0.002	-0.036	
Specification 4	reference	1.08 (0.60-1.93)	0.97 (0.57-1.67)	0.77 (0.47-1.29)	
		0.016	-0.027*	-0.016	
Base case		1.11 (0.81-1.50)	0.76 (0.57-1.03)	0.82 (0.60-1.11)	
		0.011	-0.004	-0.049	
Specification 5	reference	1.01 (0.66-1.53)	0.89 (0.60-1.32)	0.69 (0.48-1.01)	
		0.014	-0.028*	-0.015	
Base case		1.07 (0.79-1.45)	0.77 (0.57-1.03)	0.83 (0.61-1.12)	

**p-value<0.05; ***p-value<0.01; marginal effects reported in top row;
RRRs reported in bottom row with 95% CIs in parentheses.

4.5. Discussion

4.5.1. Summary of results

In the base case analysis it was seen that low income was associated with all three wheezing phenotypes, particularly persistent wheeze, in the case of the unadjusted results. Once all a priori and other explanatory variables were added to the model, there was no longer any statistically significant relationship between income and persistent wheeze or early onset wheeze. However,

a weak association remained between income and late onset wheeze (marginal effect:-0.03) although the effect was of borderline statistical significance (p-value:0.09; 95% CI:0.57, 1.03).

In the first sensitivity analysis, where the cut off for early remitting wheeze was age 7 rather than age 5, fully adjusted models showed that low income is strongly associated with persistent wheeze but not late onset wheeze. An additional sensitivity analysis where income was measured at age 9 months, as a measure of baseline income, did not show any statistically significant associations. Similarly, in the subgroup analysis where cohort children were separated into two groups according to whether or not they had a family history asthma, there were no significant associations between income and wheezing phenotypes in either subgroup.

The results suggest that a low level of family income, over the course of childhood, increases the chances that a child will suffer from late onset wheeze or persistent wheeze, which are both likely to persist into adulthood, compared to early remitting wheeze (Taylor-Robinson et al., 2015). These findings could improve the development of interventions to address the burden of disease suffered by more disadvantaged populations; interventions aimed at improving health in childhood could prevent poor health in adulthood.

4.5.2. Comparison with other studies

There have been very few studies that have investigated the relationship between income (or other measures of socioeconomic status) and wheezing phenotype showing that this dynamic way of operationalising wheeze in economic studies is novel. Previous studies support the findings from this chapter – that low socioeconomic status is associated with persistent wheeze. Using MCS data including children from age 9 months to age 7 in the UK, Taylor-Robinson et al (2015) showed that low educational achievement in mothers was associated with persistent wheeze/relapsing wheeze. Using LCA on data from children aged 6 months to 7 years, Galobardes et al (2015) identified that low paternal education (GCSE level or equivalent and

below) was associated with persistent wheeze. To my knowledge, there have been no previous studies that have shown evidence of the association between low income and late onset wheezing.

4.5.3. Other factors

In addition to income, there are other factors that were found to be associated with the different wheezing phenotypes in my analysis. Using the base case results as a point of reference, results showed that a family history of asthma was associated with all phenotypes. This is in line with previous literature which have shown evidence of a familial component to wheezing where presence of wheezing in parents increases the chances of a child having any wheezing phenotype (Rusconi et al., 1999). For the phenotype early remitting wheeze, being black Caribbean was a statistically significant determinant, which could indicate that black Caribbean children may have an underlying predisposition to wheezing compared to white children. Data from the UK has previously shown that asthma-related outcomes such as persistent wheezing, are more prevalent in certain ethnic minorities which supports the results of this chapter (Rusconi et al., 1999).

For mother and child health related factors, my results also showed that breastfeeding for four or more months significantly reduced the likelihood of early remitting wheezing. This highlights the protective nature of breastfeeding that was also seen in the cross-sectional analysis. Furthermore, Quigley et al (2018) found that prolonged breastfeeding between 6 and 9 months reduced the risk of wheezing up to age 5, in early transient wheezing but there was no clear evidence in other phenotypes. However, whether breastfeeding offers protection from conditions such as wheezing remains unresolved, especially for non-exclusive breastfeeding which is how breastfeeding is accounted for in this thesis (Azad et al., 2017; Quigley et al., 2018).

In terms of environmental factors, one factor for which there is statistically significant evidence of a reduction in the likelihood of early remitting wheezing was living in a rural area compared to an urban area. Again, there are mixed findings in the literature with respect to rural-urban morphology being associated with asthma-related outcomes but the general consensus in the literature is that wheezing is less prevalent in rural locations which is likely attributable to better air quality (Lawson et al., 2017).

Boys were more likely to have early transient, persistent and late onset wheezing compared to girls which follows the findings in previous literature that being male is a well-known risk factor for wheezing in childhood. Few studies to date have explored whether sex influences wheezing phenotypes (Deshpande and Morgan, 2016; Azad et al., 2017). The results also showed that having one or more siblings was associated with a decrease in the probability of having late onset wheeze which may support hypotheses that exposure to pathogens (transmitted by siblings) has a protective effect for younger children aged 3 and below. An explanation could be that infants exposed to other children in the home or at day care centres are at increased risk of infection from their peers, and that having infections may in turn protect against the development of asthma-related outcomes by having beneficial immunological effects in the early years as explained by the hygiene hypothesis (Azad et al., 2013). However, the results on late onset wheezing were weak and need to be interpreted cautiously.

In terms of mother and child health-related factors, results show that low birthweight was a determinant of persistent wheezing which supports evidence of low birthweight and reduced airway function in infants and children (Villamor et al., 2009). There was a also positive association of being born by c-section (emergency or planned) and persistent wheeze. C-sections are associated with long term health risks for children, such as the development of wheezing and persistence of wheezing, so this result was expected (Keag et al., 2018). Lastly, mother's mental health was significantly associated with persistent wheezing. This supports

previous evidence that poor mental health in mothers may affect their ability to provide an adequate level of care for their child, for example in managing chronic health issues, which in turn can lead to on-going health issues (Cookson et al., 2009).

4.5.4. Strengths and limitations

This chapter presents a novel approach to investigate the association between income and wheezing phenotypes in UK children. It supports a move away from dichotomous health outcomes which are commonly used in health economics' literature, to using more complex measures of wheezing outcomes. In this instance, the health outcome incorporates the element of age of onset and duration of wheezing and presents a set of phenotypes.

As mentioned for the cross-sectional analysis in chapter 3, the MCS has a number of strengths which also apply to the analysis of wheezing phenotypes. The MCS includes a large number of variables which are consistently measured at different ages, thus enabling wheezing phenotypes to be measured using data from age 3 to 11 years, and allowing the effect of income to be adjusted for a large number of confounders. Because the MCS is a large and contemporary dataset, it is likely that results can be generalisable to other high-income countries with a similar prevalence of wheezing in children. When comparing these results to previous studies that have investigated the association between socioeconomic status and wheezing phenotype, however few, the results are comparable in that low family income is associated with persistent wheezing.

The choice of wheezing phenotypes used in this thesis and their definitions used in this analysis may be open to debate as there is no standard definition in the literature. However, using a different age cut-off and redefining the boundaries of wheezing phenotype, as was done in my sensitivity analysis, offers a robustness check for my main findings and demonstrates that there can be variability in phenotypes, depending on age cut-off.

Additionally, wheezing phenotypes are based on repeated parental reports of wheezing which may not be accurate due to imperfect recall. Physiological data would strengthen the analysis but there is no physiological information in the MCS such as airflow test data, or official medical diagnostic information. Such data could make wheezing categorisation more objective and potentially more accurate. For example, the 1958 National Child Development Study (NCDS) and the 1970 British Cohort Study (BCS70) are linked to Hospital Episode Statistics (HES) data which offers objective hospital data.

4.6. Conclusion

The results of this chapter demonstrate the value in accurately classifying wheezing into longitudinal phenotypes, in order to better understand the role of low income and other factors in determining such phenotypes. Low income was shown to be a risk factor of persistent wheeze and late onset wheeze which can both lead to persistent symptoms in adulthood.

5. Panel data methods to analyse the association between family income and asthma-related outcomes

5.1. Introduction

Chapter 3 of this thesis explored the relationship between family income and asthma-related outcomes using cross-sectional analysis where data, collected at ages 9 months, 3, 5 ,7 and 11 years as part of the Millennium Study (MCS), were analysed each as a standalone dataset. The results at each age showed that there is a statistically significant relationship between low income and adverse asthma-related outcomes, however, these results were not causal.

The crux of what makes the MCS a rich and useful dataset, is its longitudinal dimension that allows observation and analysis of data from the same children, from the age of 9 months until the age of 11 years, with repeated observations made at each MCS survey age. In other words, the MCS data have both cross-sectional and time series dimensions and thus the application of statistical models that account for both of these dimensions is more complex than in a cross-sectional framework. Specific econometric methods have been developed to specifically analyse panel data (also referred to as longitudinal data). Panel data methods can be used to estimate parameters, compute partial effects and allow for valid inference when there are repeated observations for the same variables for the same study subjects (Wooldridge, 2002).

Almost all literature that has investigated the association between family income and asthma-related outcomes has done so using cross-sectional methods (as discussed in Chapter 1). One study where panel data methods were used to investigate this relationship, was Violato et al (2009), who analysed the association between family income and child respiratory disease

(asthma or wheezing) measured as a dichotomous variable using MCS data for children aged 9 months to 5 years. Fixed effects were estimated using linear probability models controlling for a range of child and parental characteristics, and the marginal effect of income was -0.012 (p-value: 0.13).

There are advantages that make using panel data methods a more robust approach than the cross-sectional one, which may allow a more accurate inference of the relationship between family income and asthma related outcomes by fully exploiting the panel dimension of the MCS. The following section discusses such advantages.

5.2. The advantage of using panel data methods

5.2.1. Unobservable variables

Cross-sectional models can theoretically control for differences in individuals, however in practice it is impossible to control for every time-invariant variable as we might not even be aware that this heterogeneity exists in the first place, and/or the relevant variables may not be available or measurable (Baltagi, 1995). Where heterogeneity is not controlled for, results run the risk of being biased, and because of this, cross-sectional analysis is likely to produce biased estimators which may lead to incorrect inference. In panel data methods, unobservable time-invariant variables are eliminated from models and unobserved heterogeneity is controlled for in this way.

5.2.2. Testing complicated behavioural hypotheses

Panel data methods take into account sequential observations of the same individuals at different time points, and incorporate this into their estimation process allowing for testing of

more complex behavioural models. The case of employment of women is a commonly used example in econometrics (Hsiao, 2014). Where, if we observed the labour market at a single point in time, we could find that the labour force participation rate in a cross-section of married women is roughly 50%, this could either be drawn from a homogenous population or it could represent a heterogeneous population where 50% of the women are from a sub-population that never worked and 50% are from a sub-population who always worked. Cross-sectional data is not able to distinguish between these two cases, but panel data can as it takes into account information from a point in time before and after, i.e. it considers sequential observations. My analysis, investigating the association between income and asthma-related outcomes over time, represent a complex model because asthma-related outcomes may vary between children from year to year and so does family income.

5.2.3. More accurate predictions and inference

Panel data methods provide more efficient econometric estimates due to the data generally having more degrees of freedom and variability compared to cross-sectional data (which is equivalent to a panel where t=1, where t represents the total number of time periods in the data) or time series data (which is comparable to a panel with n=1, where n is the total sample size) (Hsiao, 2014). This variability is because data is pooled from across both individuals and time.

5.2.4. Determinacy over the sequencing of the causal mechanism

This relates to the fact that for one variable to cause another, it must precede it in time. However, when all variables are measured at the same point in time, it is frequently not possible to establish the correct relationship and there may be issues of reverse causality. Therefore,

introducing a time dimension to the static dimension of the cross-sectional analysis allows inference of intra-individual changes over time, addressing issues of indeterminacy over sequencing (Baltagi et al., 2017).

5.3. Methodology

This section describes in detail the panel data methodology that has been applied to the MCS data. It is aimed to give a breakdown of the methods and add clarity to why they were selected for this analysis, together with their strengths and limitations. For the purposes of this thesis, I am considering two types of models – fixed and random effects models, which are commonly used in the economics literature for analysing panel data.

The decision on whether to use fixed or random effects is based on assumptions on the error component. Both estimations can be expressed by:

$$H_{it} = \beta_1 + \sum_{j=2}^{k} \beta_j X_{jit} + \alpha_i + \varepsilon_{it} \quad (5.1)$$

where H_{it} is the dependent variable observed for individual *i* at time t; X_{jit} are the observed explanatory variables, which are time-variant; and α_i represents unobserved effects that do not vary over time, for individual *i*. ε_{it} is the error term, also known as the idiosyncratic error.

5.3.1. Fixed effects methods

There are typically three different approaches to fixed effects regression analysis (Cameron and Trivedi, 2005): within-groups fixed effects; least squares dummy variable (LSDV) regression; and first differences regression. These methods are briefly discussed below, together with their advantages and limitations.

5.3.1.1. Within-groups fixed effects

In this method, the mean values of the variables for each given individual *i* are calculated equation (5.2), and subtracted from equation (5.1), resulting in equation (5.3). The mean values for individual *i*, can be specified as:

$$\bar{H}_i = \beta_1 + \sum_{j=2}^{k} \beta_j \bar{X}_{ji} + \alpha_i + \bar{\varepsilon}_i \quad (5.2)$$

Subtracting equation (5.2) from equation (5.1) gives:

$$H_{it} - \bar{H}_i = \sum_{j=2}^{k} \beta_j (X_{ijt} - \bar{X}_{ji}) + \varepsilon_{it} - \bar{\varepsilon}_i \quad (5.3)$$

One can see that the unobserved effect α_i disappears[27] once equation 5.2 is subtracted from equation 5.1. This method expresses variation from the mean of the dependent variable, $\bar{H}$, in terms of variation from the means of explanatory variables, $\bar{X}_{ij}$ (Wooldridge, 2002; Cameron and Trivedi, 2005). It provides a way of tackling the issue of unobserved heterogeneity, however there are some limitations. Firstly, the intercept, β_1 drops out of the model during the subtraction as well as any X that is constant over all time periods i.e. $(X_{ijt} - \bar{X}_{ji})$=0 , which in essence means that coefficients of time-invariant variables cannot be estimated. Secondly, the precision of the error term depends on the mean square deviation of explanatory variables to be high compared to the variance of the error term. Where the variation in $X_{ijt} - \bar{X}_{ji}$ is small compared to the variation in the error term, this may give rise to imprecise estimates (Wooldridge, 2002).

[27] α_i disappears from the equation because $\bar{\alpha}_i = T^{-1} \sum_{t=1}^{T} \alpha_i = T^{-1} T \alpha_i = \alpha_i$

5.3.1.2. The dummy variable regression or least squares dummy Variable (LSDV) fixed effects

In this method, the unobserved effect is explicitly introduced into the model as a parameter to be estimated for each individual *i*. A dummy variable A_i is defined for each cross-sectional observation (i.e. for each individual) *i* , which is equal to 1 when it refers to individual *i* and 0 otherwise. Equation (5.1) can then be written as:

$$H_{it} = \sum_{j=2}^{k} \beta_j X_{jit} + \sum_{i=1}^{n} \alpha_i A_i + \varepsilon_{it} \quad (5.4)$$

In equation (5.4), the unobserved effect α_i for each individual *i* is expressed as the coefficient of dummy variable A_i, and the term $\alpha_i A_i$ represents an unobserved effect on the dependent variable H_i. The model is then fitted using OLS. One individual is defined as the reference category, but the choice of the reference is often arbitrary and thus the interpretation of α_i varies depending on the reference category. Another limitation is that, if there is a large number of individuals in the sample, this method is not very practical given the high number of dummy variables that would need to be included in the model. It has been shown that the LSDV methods yield the same results as the within-groups FE estimator described previously, as it has a similar methodological approach. And therefore the same advantages and limitations are applicable (Wooldridge, 2002; Cameron and Trivedi, 2005).

5.3.1.3. First differences fixed effects

In the first differences approach, the unobserved effect, α_i ,is eliminated by subtracting the model at time *t-1* from the model at time *t* (Wooldridge, 2002; Cameron and Trivedi, 2005). The model at time *t* is as specified in equation (5.1), the same model at time *t-1* can be expressed as:

$$H_{it-1} = \beta_1 + \sum_{j=2}^{k} \beta_j X_{jit-1} + \alpha_i + \varepsilon_{it-1} \quad (5.5)$$

Subtracting equation (5.5) from (5.1), this results in:

$$\Delta H_{it} = \sum_{j=2}^{k} \beta_j \Delta X_{jit} + \varepsilon_{it} - \varepsilon_{it-1} \quad (5.6)$$

It can be seen that the unobserved heterogeneity α_i is no longer present in equation (5.6). The same limitation as described in the previous two fixed effects methods (within-groups fixed effects and LSDV regression) still apply here – the intercept and any explanatory variables that are time-invariant will be eliminated from the model, with only estimations of time-variant explanatory variables being obtained.

5.3.1.4. Selecting the most appropriate estimator

In deciding which of the above methods should be used, a test for serial correlation in the idiosyncratic error term (ε_{it}), was performed (Wooldridge, 2002). As mentioned, the LSDV approach and within-groups fixed-effects (FE) approach yield the same estimator, therefore a choice needs to be made between either of these and the first differences approach. When t=2, the within-groups and first differencing estimates are identical, but they differ once t>3 and a choice has to be made (Wooldridge, 2002). In the presence of serial correlation in the idiosyncratic error term, within-groups FEs is more suitable. The test for serial correlation involved the command 'xtserial' in Stata. The test for serial correlation in the first differences estimator had a p-value<0.001[28] which means that there is serial correlation in the idiosyncratic

[28] The xtserial command in Stata was used to assess serial correlation. The null hypothesis of the test is: no serial correlation and the alternative hypothesis is the presence of serial correlation of ε_{it}.

error term, suggesting that the alternative within-groups fixed effects estimator would produce a more efficient estimator (Wooldridge, 2002).

5.3.2. Random effects

As was seen with the different fixed effects approaches, explanatory variables that are time-invariant get dropped from the model, therefore no inference can be made from these variables as their coefficients are not estimated; whether they are important determinant factors would remain unknown. Randoms effects (RE) models offer a solution to this limitation of FE models. However, two conditions must be fulfilled in order for the RE approach to be used. The error term ε_{it} in equation (5.1) can be combined with the unobserved effects α_i and be expressed collectively, resulting in a modified equation:

$$H_{it} = \beta_1 + \sum_{j=2}^{k} \beta_j X_{jit} + (\alpha_i + \varepsilon_{it}) \quad (5.7)$$

where:

$$u_{it} = \alpha_i + \varepsilon_{it} \quad (5.8)$$

and therefore:

$$H_{it} = \beta_1 + \sum_{j=2}^{k} \beta_j X_{jit} + u_{it} \quad (5.9)$$

The unobserved effect added to the idiosyncratic error term creates u_{it}, a composite error term (Wooldridge, 2002; Cameron and Trivedi, 2005; Hsiao, 2014). In light of this composite error, the two assumptions that are imposed by RE are firstly, one of strict exogeneity:

$$E(u_{it}|x_i, \alpha_i) = 0, t = 1, \dots, T.$$

This implies that each unobserved explanatory variable is drawn randomly from the given distribution, which in turn implies that explanatory variables are uncorrelated with the idiosyncratic error in each time period. The second condition is that of orthogonality:

$$E(\alpha_i|x_i) = E(\alpha_i) = 0$$

The second condition implies zero correlation between the observed explanatory variables x_i and the unobserved effects α_i, meaning α_i is independent of x_i.

5.3.3. Choosing between fixed effects and random effects

To decide between the FE and RE models, both models are estimated then the Hausman test is applied. The null hypothesis of the test states that the conditional expectation of the unobserved effects (α_i) is zero given X_i, the observable explanatory variables[29]. If the null hypothesis holds, the RE and FE estimators are both consistent estimators and the RE estimator would then be preferred, as it is more efficient and estimates both time-invariant and time-varying determinants. If the null hypothesis is rejected, then it can be concluded that the RE estimator is not a suitable estimator as the condition $E(\alpha_i|x_i) = E(\alpha_i) = 0$ is violated. In this case the FE estimator is favourable (Wooldridge, 2002; Cameron and Trivedi, 2005; Hsiao, 2014). The null (H_0) and alternative hypotheses (H_1) of the Hausman test can be expressed as:

$$H_0: \mathrm{E}(\alpha_i|X_i) = 0$$

$$H_1: \mathrm{E}(\alpha_i|X_i) \neq 0$$

[29] Alternatively, the null hypothesis can be stated as the covariance between α_i and X_i being equal to zero.

In this chapter, the Hausman test will be used after estimating both FEs and REs to select which estimation models gives consistent and efficient estimators (Cameron and Trivedi, 2005).

5.3.4. Theoretical model

Similarly to the theoretical approach used in the cross-sectional analysis in Chapter 3, and the multinomial phenotype analysis in Chapter 4, child asthma-related outcomes are estimated using the health capital model developed by Grossman (1972a, 1972b and 2000). To recap, equation (5.12) below is the child-health production function and includes family income, parental and child characteristics, and household and environmental factors:

$$H_{it}^{c} = \delta + \beta lnY_{it} + \gamma X_{it}^{p} + \theta X_{it}^{C} + \alpha_i + \varepsilon_{it} \quad (5.12)$$

where:

H_{it}^{c} is child *i*'s asthma-related outcome at time *t*, Y_{it} is family income for child *i* at time *t*; X_{it}^{p} is a set of parental characteristics; X_{it}^{C} is a set of child characteristics; α_i represents unobservable, time-invariant variables; and ε_{it} is the idiosyncratic error. The latter two expressions combined make the composite error *u*.

5.3.5. Data

The same child asthma-related outcomes used in cross-sectional analysis (Chapter 3) and analysis of wheezing phenotypes (Chapter 4) are used in this chapter. To recap, these are ever asthma, wheezing in the last 12 months, and asthma medication use. Table 5.1 reports which of the three outcomes are available at each survey (child ages 3, 5, 7 and 11 years). The natural logarithm of current family income in (2011 prices) is the measure of family income (Chapter

2, Section 2.8 and 2.9). Time-invariant and time-variant factors that are added to the model as confounders. Confounders included a priori factors, socioeconomic factors, mother and child health-related factors and household and environmental factors and are described in Appendix D-1.

Table 5.1: Asthma-related outcomes at each child age

Outcome	Age 3 (t=1)	Age 5 (t=2)	Age 7 (t=3)	Age 11 (t=4)
Ever asthma (Yes/No)	✓	✓	✓	✓
Wheezing in the last 12 months (Yes/No)	✓	✓	✓	✓
Asthma medication use(Yes/No)	N/A	✓	✓	✓

5.3.6. Modelling approach

In Stata, panel data commands (xt) for estimating FE and RE cannot be used with survey data syntax (svy) due to software limitations. Therefore I have tried different approaches to deal with this limitation and compared the results. The approaches I used to overcome the above limitation in Stata are:

- Approach 1: Conventional panel data methods as discussed above (FE and RE estimations). Firstly, these estimations were made using the command 'xtreg', which accounts for the longitudinal nature of the data but does not take into account survey properties of the MCS, such as survey weights (I refer to models hereon as 'weighted' and 'unweighted'). The 'xtreg' command estimates linear probability models (LPM) with coefficients that are directly interpretable as probabilities. With my data, the command

'xtlogit'[30] which is more conventionally used with categorical outcomes, yields a high number of dropped observations due to instances where a health outcome was either yes or no (1 or 0) for all survey ages[31]. For unweighted models, the Hausman test was used to decide on appropriateness of models[32]. Weighted models were also estimated. For the FE models, survey weights were applied as options in the Stata syntax as an alternative to the 'svy' command, which cannot be used with 'xt' syntax. For RE models, survey weights cannot be added, so I used the closely related maximum likelihood estimation (MLE) command, which does allow for survey weights. The MLE is a random effects model estimated using maximum likelihood rather than generalized method of moments (GMM) estimator used in RE (Stata, 2020). Unadjusted models were first estimated for unweighted FE and RE models and weighted FE and MLE, with family income as the only determinant factor. Fully adjusted models for were then estimated with a full list of variables[33].

- Approach 2: an alternative ad hoc approach was devised, which allowed me to account for both the complex survey design and the longitudinal nature of the data, using the Stata 'svy' command to allow for clustering by electoral ward and child. Single regression models are estimated for each asthma-related outcome with an interaction term between family income and MCS survey age[34]. In this way the overall association between income and outcome is estimated at each MCS survey age. Unadjusted estimations were first

[30] As with xtreg, xtlogit also cannot be used with the svy specification in Stata.

[31] Models were run using xtlogit and marginal effects derived to ensure that the results from LPMs (using xtreg) were comparable. See Appendix D-3.

[32] The Hausman test cannot be applied to models that use weights.

[33] Variables included in the analysis can be found in Appendix D-1.

[34] The time variable corresponds to each survey of the MCS, i.e. sweep 2, 3, 4 and 5

made with only family income interacting with age as a determinant, and then a full set of determinant factors. Estimations were made using the 'svylogit' command in Stata and then marginal effects of income were estimated at each age using the 'margins' command.

Each of the above analysis was repeated for each of the three asthma-related outcomes: Ever asthma, wheezing in the last 12 months, and asthma medication use. Family income was included in models as the natural logarithm of current transient income (Chapter 2, Section 2.9).

5.4. Results

This section describes the summary statistics for family income and asthma-related outcomes in the MCS for combined data from MCS 2, 3, 4 and 5. Summary statistics for panel data were generated using 'xtsum' (for income) and 'xttab' for asthma-related outcomes (Cameron and Trivedi, 2010). Results from Approach 1 and 2 are then described.

Table 5.2: Longitudinal summary statistics of income and asthma-related outcomes

Summary Statistics	Ln of equivalised family income	Ever asthma	Wheezing in the last 12 months	Asthma medication use[35]
Mean Standard Deviation/ Frequency[36]	9.90			
Overall	0.56	15.13	14.8	4.57
Between	0.55	24.86	33.77	8.91
Within	0.10	61.23	43.82	51.28
Min	7.29			
Max	11.36			

[35] Time periods for asthma medication use is equal to 3 (compared to 4 for other outcomes) as this data was only available at MCS 3, 4 and 5.

[36] For the dichotomous asthma-related health outcomes, the values for overall, between and within are % frequencies; for income standard deviations are reported.

Summary Statistics	Ln of equivalised family income	Ever asthma	Wheezing in the last 12 months	Asthma medication use[35]
Sample size (N)	10,260			
Time periods (T)	4(or 3)			

Table 5.2 shows that out of the sample of 10,260 families, the natural logarithm of mean equivalised family income is 9.9. The between variation in income, i.e. the time-invariant variation across children, is 0.55 and the within variation, i.e. the time-variant variation, is 0.10. This indicates that there is a lot more variation in income across individual children, compared to the same child across survey periods.

For asthma-related outcomes, summary statistics indicate that 15% of all responses to ever asthma, across the pooled sample, are yes. The between figure indicates that 25% of parents reported that their child had ever had asthma in at least one survey. The within figure indicates that of those who had ever asthma at least once, the proportion of times they are in this state was 61% (on average). For wheezing in the last 12 month, 15% of all responses across the pooled sample, are yes; 34% reported wheezing in the last 12 months at least once and are in this state 44% of the time. For asthma medication use, 5% responses to asthma medication use across the pooled sample, are yes; 9% of the sample reported asthma medication use at least once and are in this state 51% of the time.

5.4.1. Approach 1: Using panel data methods

The estimates for the unadjusted FE and RE models for all asthma-related outcomes are displayed in Table 5.3 (unweighted) and Table 5.4 (weighted). In the unweighted FE model for the outcome ever asthma, the marginal effect of income on ever asthma is -0.015 (p-value: 0.01) indicating that a 1% increase in income was associated with a decrease in the probability of a

child ever having asthma of -0.015. The corresponding figure for the FE model with weights is -0.014 and is also statistically significant (p-value:0.01). In the unadjusted and unweighted RE model, a 1% increase in income was associated with a decrease in the probability of ever asthma of 0.001 (Table 5.3), the corresponding weighted MLE value is -0.003 (Table 5.4) and neither are statistically significant. For the unweighted models, the Hausman test indicates that the FE is preferred to RE (p-value<0.01).

For wheezing in the last 12 months, the unadjusted unweighted FE model shows that a 1% increase in income was associated with a statistically significant decrease in the probability of a child having wheezing in the last 12 months of 0.019 (p-value:0.001). In the unadjusted weighted FE model the corresponding coefficient was -0.03 (p-value: 0.001). In the unadjusted unweighted RE model, the decrease in the probability of wheezing in the last 12 months was -0.023 (p-value: 0.001) and in the weighted MLE -0.026 (p-value:0.001). Comparing the unweighted models, the RE is preferred to the FE on the basis of the Hausman test (p-value: 0.12) (Table 5.3). For the outcome asthma medication use, both unadjusted weighted and unweighted FE model estimations are not statistically significant. Unweighted RE and weighted MLE models did not yield any statistically significant results either in unadjusted models (Table 5.3 and 5.4).

Table 5.3: Unadjusted FE and RE results (unweighted)

	Ever asthma		**Wheezing in the last 12 months**		**Asthma medication use**	
	FE	**RE**	**FE**	**RE**	**FE**	**RE**
Ln Income	-0.015***	-0.001	-0.019***	-0.023***	0.004	-0.003
	[-0.009;-0.021]	[-0.007;0.004]	[-0.027;-0.011]	[-0.029;-0.017]	[-0.002;0.009]	[-0.007;0.002]
Hausman (p-value)	<0.001		0.115		<0.001	

* p<0.10, **p<0.05, ***p<0.01; Standard errors in parentheses

Table 5.4: Unadjusted regression results, (weighted)

	Ever Asthma		Wheezing in the last 12 months		Asthma medication use	
	FE	**MLE**	**FE**	**MLE**	**FE**	**MLE**
Ln Income	0.014***	-0.003	-0.025***	-0.026***	0.003	-0.002
	[-0.006;-0.022]	[-0.008;0.002]	[-0.035;-0.014]	[-0.032;-0.021]	[-0.004;0.010]	[-0.006;0.002]

* p<0.10, **p<0.05, ***p<0.01; Standard errors in parentheses

Tables 5.5 shows the fully adjusted FE models (both unweighted and weighted models) and Table 5.6 the fully adjusted unweighted RE and weighted MLE models for the outcome ever asthma, the former populated with time-varying determinants and the latter with both time-varying and time invariant determinants. Tables 5.7 and 5.8 report corresponding results for wheezing in the last 12 months, and Tables 5.9 and 5.10 for asthma medication use.

For the ever asthma, in the unweighted FE model a 1% increase in family income was associated with a decrease in the probability of ever asthma of 0.008 (p-value: 0.13), but this association was very weak (Table 5.5). The corresponding marginal effect for the weighted FE model was -0.007 (p-value: 0.24). In the unweighted FE model, mother's general health was weakly associated with ever asthma, with fair or poor health increasing the probability of ever asthma in children by 0.01 compared to mothers in good or excellent health (p-value: 0.10) and no other variables were statistically significant apart from child age with a marginal effects of 0.0002 (p-value: 0.001). In the weighted FE model, the association of housing on ever wheeze was significant with children living in council housing being more likely to be reported to ever having asthma compared to children in mortgaged or owned homes, with marginal effect 0.02 (p-value:0.02). Child age was also statistically significant with identical association to the unweighted model.

In both of the unweighted RE and weighted MLE models, the marginal effect of income on ever asthma was the same, 0.011 (p-value: 0.001) (Table 5.6). Time-invariant factors that were associated with ever asthma were child sex, family history of asthma, birthweight, breastfeeding, smoking during pregnancy and damp in the home at 9 months. These factors were statistically significant in both the RE and the MLE models. Time-variant factors that were associated with ever asthma are mother's general health, in both models. When selecting between the FE and RE model for ever asthma, a Hausman test between the non-weighted adjusted FE and RE models with a p-value of <0.01, indicated that the RE is not a consistent estimator, the FE model is preferable in this case.

Table 5.5: Adjusted FE - Ever asthma

	FE (unweighted)		**FE (weighted)**	
	Coeff	**95% CI**	**Coeff**	**95% CI**
Ln income	-0.008*	[-0.016,0.001]	-0.007	[-0.016,0.002]
A priori factors				
Child age	0.0002***	[0.0002,0.0003]	0.0002***	[0.0002,0.0003]
Housing tenure				
Mortgaged or owned	-	-	-	-
Rented privately	0.014	[-0.335,0.362]	0.015	[-0.023,0.052]
Council housing	0.021	[-0.327,0.369]	0.018**	[0.001,0.035]
Mother's general health				
Good or excellent health	-	-	-	-
Fair or poor health	0.011*	[-0.0001,0.023]	0.013	[-0.004,0.031]
Mother's mental health				
No or low distress	-	-	-	-
Medium distress	0.004	[-0.005,0.013]	0.002	[-0.008,0.012]
High distress	0.000	[-0.022,0.022]	-0.012	[-0.047,0.022]

* p<0.10, **p<0.05, ***p<0.01

Table 5.6: Adjusted RE and MLE - Ever asthma

	RE (unweighted)		**MLE (weighted)**	
	Coeff	**95% CI**	**Coeff**	**95% CI**
Ln income	-0.011***	[-0.018,-0.004]	-0.011***	[-0.018,-0.004]
A priori factors				
Child age	0.0002	[0.0002,0.0003]	0.0002	[0.002,0.003]
Ethnicity				
White	-	-	-	-
Mixed	-0.014	[-0.085,0.057]	0.065	[0.005,0.126]
Indian	-0.002	[-0.043,0.040]	-0.005	[-0.048,0.038]
Pakistani	-0.006	[-0.043,0.031]	0.0001	[-0.038,0.038]
Bangladeshi	-0.072	[-0.135,-0.008]	-0.072	[-0.143,-0.001]
Black Caribbean	0.050	[-0.013,0.113]	0.048	[-0.009,0.104]
Black African	0.035	[-0.024,0.095]	0.012	[-0.041,0.065]
Other	-0.009	[-0.061,0.044]	-0.034	[-0.084,0.017]
History of family asthma	0.115***	[0.101,0.128]	0.119***	[0.106,0.131]
Child sex	0.043***	[0.031,0.055]	0.046***	[0.035,0.057]
Birthweight				
Normal	-	-	-	-
Low	0.048***	[0.020,0.075]	0.048***	[0.023,0.073]
Very low	0.140***	[0.071,0.210]	0.180***	[0.119,0.241]
Mother's age				
14-19	-	-	-	-
20-24	0.005	[-0.024,0.035]	0.021	[-0.004,0.046]
25-29	-0.015	[-0.044,0.013]	0.000	[-0.024,0.024]
30-34	-0.028	[-0.057,0.001]	-0.010	[-0.035,0.015]
35-39	-0.031	[-0.063,0.001]	-0.008	[-0.035,0.020]
>=40	-0.037	[-0.083,0.009]	-0.033	[-0.076,0.009]
Mother's education				
NVQ level 1	-	-	-	-
NVQ level 2	0.001	[-0.023,0.026]	-0.005	[-0.027,0.016]
NVQ level 3	-0.015	[-0.042,0.012]	-0.024	[-0.048,0.001]
NVQ level 4	0.004	[-0.022,0.030]	-0.003	[-0.026,0.020]
NVQ level 5	-0.002	[-0.040,0.036]	0.0001	[-0.037,0.037]
Overseas	0.009	[-0.039,0.056]	0.014	[-0.028,0.056]
None of these	0.011	[-0.018,0.040]	-0.011	[-0.037,0.014]
Housing tenure				
Mortgaged or owned	-	-	-	-
Rented privately	0.064	[-0.271,0.399]	0.060	[-0.158,0.278]
Council housing	0.05	[-0.284,0.385]	0.047	[-0.171,0.264]
Breast feeding				

	RE (unweighted)		MLE (weighted)	
	Coeff	**95% CI**	**Coeff**	**95% CI**
Never breast fed	-	-	-	-
<2 months	-0.014*	[-0.030,0.002]	-0.009	[-0.024,0.006]
2-4 months	-0.029***	[-0.050,-0.009]	-0.022**	[-0.042,-0.003]
4-6 months	-0.022*	[-0.046,0.002]	-0.012	[-0.035,0.010]
>6 months	-0.041***	[-0.059,-0.022]	-0.041***	[-0.059,-0.024]
Delivery mode				
Normal	-0.101	[-0.293,0.091]	-0.035	[-0.201,0.132]
Assisted	-0.095	[-0.288,0.097]	-0.019	[-0.186,0.148]
Planned c-section	-0.081	[-0.274,0.111]	-0.015	[-0.183,0.152]
Emergency c-section	-0.084	[-0.277,0.108]	-0.010	[-0.177,0.157]
Alcohol in pregnancy				
Never	-	-	-	-
Light	-0.013	[-0.028,0.001]	-0.021	[-0.034,0.008]
Moderate	-0.030	[-0.058,0.003]	-0.021	[-0.046,0.004]
Heavy	0.025	[-0.018,0.068]	0.009	[-0.030,0.048]
Smoking in pregnancy				
Never	-	-	-	-
Gave up	0.015	[-0.004,0.034]	0.009	[-0.009,0.026]
Kept smoking	0.021**	[0.004,0.039]	0.021***	[0.006,0.037]
Mother's BMI				
Underweight	-	-	-	-
Normal	-0.001	[-0.030,0.028]	0.006	[-0.020,0.033]
Overweight	0.008	[-0.023,0.039]	0.014	[-0.015,0.042]
Obese	-0.006	[-0.038,0.026]	0.005	[-0.024,0.034]
Mother's general health				
Good or excellent health	-	-	-	-
Fair or poor health	0.018***	[0.007,0.028]	0.020***	[0.011,0.030]
Mother's mental health				
No or low distress	-	-	-	-
Medium distress	0.006	[-0.002,0.014]	0.003	[-0.004,0.010]
High distress	0.013	[-0.007,0.033]	0.003	[-0.015,0.020]
Smoking around child	-0.012	[-0.032,0.008]	-0.003	[-0.020,0.015]
Damp	0.016*	[-0.002,0.035]	0.015*	[-0.002,0.032]
Fume exposure	0.007	[-0.005,0.020]	0.022	[-0.009,0.024]
Rural-Urban morphology				
Urban	-	-	-	-
Mixed Urban-Rural	-0.001	[-0.021,0.020]	-0.013	[-0.032,0.006]
Rural	-0.010	[-0.029,0.008]	-0.015	[-0.033,0.003]
Hausman p-value	<0.01			

* p<0.10, **p<0.05, ***p<0.01

Table 5.7 shows the adjusted weighted and unweighted FE models for wheezing in the last 12 months. In both models the estimated association between income and wheezing in the last 12 months was not statistically significant. Child age was negatively associated with wheezing in the last 12 months for both models, but the effects are very small with marginal effect -0.001 (p-value: 0.001).

Table 5.7: Adjusted FE - Wheezing in the last 12 months

	FE (unweighted)		**FE (weighted)**	
	Coeff	**95% CI**	**Coeff**	**95% CI**
Ln income	0.009	[-0.002,0.021]	0.006	[-0.009,0.021]
A priori factors				
Child age	-0.0005***	[-0.0004,-0.0005]	-0.0005***	[-0.0004,-0.0005]
Housing tenure				
Mortgaged or owned	-	-	-	-
Rented privately	-0.025	[-0.519,0.470]	-0.057	[-0.097,0.017]
Council housing	-0.045	[-0.539,0.449]	-0.052	[-0.071,0.032]
Mother's health				
Good or excellent health	-	-	-	-
Fair or poor health	0.012	[-0.005,0.029]	0.01	[-0.013,0.033]
Mother's mental health				
No or low distress	-	-	-	-
Medium distress	-0.007	[-0.020,0.005]	-0.005	[-0.021,0.012]
High distress	-0.015	[-0.046,0.017]	-0.011	[-0.056,0.034]

* p<0.10, **p<0.05, ***p<0.01

The association between wheezing in the last 12 months and income was not statistically significant in neither the adjusted RE and MLE models (Table 5.8). Time-invariant determinants that were statistically significant in both RE and MLE models, increasing the probability of wheezing, are ethnicity, (black Caribbean), birthweight (low or very low), mother's age at time of birth (>40 years) and smoking during pregnancy. Time-varying covariates that were statistically significant determinants increasing the probability of wheezing

in the last 12 months include mother's general health (fair or poor health) and mothers with medium or high mental health distress levels. In both models, breastfeeding for two or more months reduced the probability of wheezing in the last 12 months. The Hausman test to select between unweighted FE and RE models indicates the FE model is preferred (p-value<0.01) (Table 5.8).

Table 5.8: Adjusted RE and MLE - Wheezing in the last 12 months

	RE (unweighted)		**MLE (weighted)**	
	Coeff	**95% CI**	**Coeff**	**95% CI**
Ln income	0.001	[-0.008,0.010]	-0.003	[-0.011,0.006]
A priori factors				
Child age	-0.0005***	[-0.0006,-0.0004]	-0.0005***	[-0.0006,-0.0005]
Ethnicity				
White	-	-	-	-
Mixed	-0.019	[-0.083,0.046]	0.045	[-0.009,0.100]
Indian	0.026	[-0.013,0.064]	0.025	[-0.015,0.065]
Pakistani	0.021	[-0.014,0.056]	0.015	[-0.020,0.051]
Bangladeshi	-0.054	[-0.115,0.006]	-0.038	[-0.105,0.029]
Black Caribbean	0.053*	[-0.004,0.111]	0.063**	[0.011,0.115]
Black African	-0.023	[-0.078,0.033]	-0.034	[-0.083,0.016]
Other	-0.045	[-0.094,-0.004]	-0.056	[-0.103,0.009]
History of family asthma	0.091***	[0.079,0.104]	0.089***	[0.077,0.100]
Child sex	0.048***	[0.037,0.059]	0.049***	[0.039,0.059]
Birth weight				
Normal	-	-	-	-
Low	0.045***	[0.020,0.070]	0.047***	[0.024,0.070]
Very low	0.109***	[0.046,0.173]	0.106***	[0.051,0.161]
Mother's age				
14-19	-	-	-	-
20-24	0.024	[-0.003,0.051]	0.027	[-0.005,0.050]
25-29	0.020	[-0.006,0.046]	0.031	[-0.010,0.053]
30-34	0.012	[-0.015,0.039]	0.031	[-0.008,0.053]
35-39	0.013	[-0.016,0.042]	0.032**	[0.007,0.057]
>=40	0.054**	[0.012,0.095]	0.070***	[0.033,0.108]
Mother's education				
NVQ level 1	-	-	-	-
NVQ level 2	0.007	[-0.015,0.029]	0.006	[-0.013,0.026]
NVQ level 3	-0.01	[-0.035,0.015]	-0.009	[-0.031,0.013]
NVQ level 4	0.009	[-0.015,0.033]	0.01	[-0.011,0.031]
NVQ level 5	0.01	[-0.024,0.045]	0.026	[-0.008,0.059]

	RE (unweighted)		MLE (weighted)	
	Coeff	**95% CI**	**Coeff**	**95% CI**
Overseas	0.008	[-0.035,0.052]	0.009	[-0.029,0.048]
None of these	0.009	[-0.018,0.036]	-0.001	[-0.024,0.022]
Housing tenure				
Mortgaged or owned	-	-	-	-
Rented privately	0.034	[-0.418,0.485]	0.025	[-0.267,0.318]
Council housing	0.017	[-0.435,0.468]	0.004	[-0.288,0.296]
Breastfeeding				
Never breast fed	-	-	-	-
<2 months	-0.014	[-0.029,0.001]	-0.014**	[-0.027,-0.001]
2-4 months	-0.030***	[-0.049,-0.012]	-0.024***	[-0.042,-0.007]
4-6 months	-0.021*	[-0.043,0.001]	-0.020*	[-0.040,0.001]
>6 months	-0.026***	[-0.043,-0.009]	-0.024***	[-0.040,-0.009]
Delivery mode				
Normal	-0.058	[-0.236,0.120]	0.020	[-0.134,0.173]
Assisted	-0.049	[-0.228,0.130]	0.037	[-0.116,0.191]
Planned c-section	-0.04	[-0.219,0.139]	0.038	[-0.116,0.192]
Emergency c-section	-0.036	[-0.214,0.143]	0.049	[-0.104,0.203]
Alcohol in pregnancy				
Never	-	-	-	-
Light	-0.005	[-0.018,0.008]	-0.011	[-0.022,0.001]
Moderate	-0.029	[-0.054,0.004]	-0.020	[-0.043,0.002]
Heavy	-0.005	[-0.044,0.034]	-0.002	[-0.037,0.032]
Smoking in pregnancy				
Never	-	-	-	-
Gave up	0.011	[-0.006,0.028]	0.011	[-0.004,0.027]
Kept smoking	0.017**	[0.001,0.033]	0.017**	[0.003,0.031]
Mother's BMI				
Underweight	-	-	-	-
Normal	-0.007	[-0.033,0.020]	0.003	[-0.020,0.026]
Overweight	0.010	[-0.018,0.038]	0.023	[-0.002,0.049]
Obese	0.000	[-0.029,0.029]	0.014	[-0.012,0.040]
Mother's health				
Good or excellent health	-	-	-	-
Fair or poor health	0.025***	[0.011,0.038]	0.023***	[0.011,0.035]
Mother's mental health				
No or low distress	-	-	-	-
Medium distress	0.014***	[0.005,0.024]	0.017***	[0.008,0.026]
High distress	0.037***	[0.012,0.062]	0.043***	[0.021,0.065]
Smoking around child	-0.003	[-0.021,0.015]	0.002	[-0.014,0.018]
Damp	0.013	[-0.004,0.030]	-0.003	[-0.018,0.012]
Fume exposure	0.001	[-0.011,0.012]	0.013*	[0.003,0.024]
Rural-Urban morphology				

	RE (unweighted)		MLE (weighted)	
	Coeff	**95% CI**	**Coeff**	**95% CI**
Urban	-	-	-	-
Mixed Urban-Rural	-0.009	[-0.028,0.009]	-0.012	[-0.029,0.005]
Rural	-0.008	[-0.024,0.009]	-0.001	[-0.017,0.015]
Hausman p-value	<0.01			

* p<0.10, **p<0.05, ***p<0.01

For asthma medication use, both weighted and unweighted FE models did not show a statistically significant relationship between income and asthma medication use (Table 5.9). No other time-variant determinants had a statistically significant association with asthma medication use.

Table 5.9: Adjusted FE - Asthma medication use

	FE (unweighted)		FE (weighted)	
	Coeff	**95% CI**	**Coeff**	**95% CI**
Ln income	-0.004	[-0.012,0.005]	-0.001	[-0.013,0.012]
A priori factors				
Child age	0.0001	[-0.0001,0.0003]	0.0001	[-0.0001,0.0002]
Housing tenure				
Mortgaged or owned	-	-	-	-
Rented privately	0.013	[-0.270,0.296]	0.000	[-0.037,0.036]
Council housing	0.002	[-0.280,0.283]	0.002	[-0.014,0.019]
Mother's health				
Good or excellent health	-	-	-	-
Fair or poor health	0.009	[-0.003,0.021]	0.006	[-0.010,0.023]
Mother's mental health				
No or low distress	-	-	-	-
Medium distress	0.004	[-0.005,0.013]	0.003	[-0.009,0.016]
High distress	0.016	[-0.006,0.038]	0.020	[-0.011,0.051]

* p<0.10, **p<0.05, ***p<0.01

Table 5.10 displays the fully adjusted RE and MLE models results for the outcome asthma medication use. In the unweighted RE model, a 1% increase in income was associated with a decrease in the probability of asthma medication use (marginal effect: -0.006; p-value: 0.02). Likewise in the weighted MLE model, the corresponding marginal effect was -0.01 (p-value:

0.005). Time-invariant factors that were positively associated with asthma medication use in both RE and MLE models were low birthweight, a male child and family history of asthma. Breastfeeding for two or more months was negatively associated with asthma medication use but these results were only significant in the RE model. Time-variant factors that were positively associated with asthma medication use are mother's general health and mental health, where fair or poor general health, and high distress levels were positive determinants of asthma medication use in both RE and MLE models. The Hausman test for the unweighted models shows that the RE provides a consistent and efficient estimator (p-value: 0.43) (Table 5.10)[37].

Table 5.10: Adjusted RE and MLE - Asthma medication use

	RE (unweighted)		**MLE (unweighted)**	
	Coeff	**95% CI**	**Coeff**	**95% CI**
Ln income	-0.006**	[-0.012,-0.0001]	-0.005*	[-0.011,-0.0001]
A priori factors				
Child age	0.0001***	[0.0002,0.0002]	0.0002***	[0.0002,0.0003]
Ethnicity				
White	-	-	-	-
Mixed	0.009	[-0.032,0.050]	0.019	[-0.015,0.054]
Indian	0.007	[-0.017,0.031]	0.007	[-0.018,0.032]
Pakistani	-0.005	[-0.027,0.018]	-0.006	[-0.028,0.016]
Bangladeshi	-0.019	[-0.057,0.018]	-0.020	[-0.062,0.021]
Black Caribbean	0.000	[-0.036,0.037]	-0.002	[-0.035,0.031]
Black African	0.004	[-0.031,0.039]	-0.013	[-0.044,0.018]
Other	-0.021	[-0.051,0.009]	-0.028	[-0.057,0.001]
History of family asthma	0.040***	[0.032,0.048]	0.036***	[0.029,0.043]
Child sex	0.015***	[0.009,0.022]	0.014***	[0.007,0.020]
Birthweight				
Normal	-	-	-	-
Low	0.029***	[0.014,0.045]	0.036***	[0.022,0.050]
Very low	0.026	[-0.014,0.067]	0.048***	[0.013,0.083]

[37] Approach 1 results using xtlogit were comparable to xtreg results for all outcomes ever asthma, wheezing in the last 12 months and asthma medication use. Results for unadjusted models are reported in Appendix D-3.

	RE (unweighted)		MLE (unweighted)	
	Coeff	**95% CI**	**Coeff**	**95% CI**
Mother's age				
14-19	-	-	-	-
20-24	0.025	[-0.007,0.042]	0.031	[-0.017,0.045]
25-29	0.022	[-0.005,0.039]	0.029	[-0.015,0.043]
30-34	0.021	[-0.006,0.040]	0.027	[-0.013,0.042]
35-39	0.022	[-0.004,0.041]	0.029	[-0.014,0.045]
>=40	0.009	[-0.018,0.035]	0.017	[-0.007,0.041]
Mother's education				
NVQ level 1	-	-	-	-
NVQ level 2	0.011	[-0.004,0.025]	0.008	[-0.005,0.020]
NVQ level 3	0.008	[-0.008,0.024]	0.007	[-0.007,0.021]
NVQ level 4	0.020	[-0.005,0.036]	0.021	[-0.008,0.034]
NVQ level 5	0.025	[-0.003,0.047]	0.035	[-0.013,0.056]
Overseas	0.006	[-0.022,0.033]	0.026	[0.001,0.050]
None of these	0.018	[-0.001,0.035]	0.015	[-0.000,0.029]
Housing tenure				
Mortgaged or owned	-	-	-	-
Rented privately	0.02	[-0.222,0.263]	0.015	[-0.143,0.173]
Council housing	0.013	[-0.229,0.255]	0.009	[-0.148,0.167]
Breast feeding				
Never breast fed	-	-	-	-
<2 months	-0.005	[-0.015,0.004]	0.001	[-0.007,0.010]
2-4 months	-0.013**	[-0.024,-0.001]	-0.004	[-0.015,0.007]
4-6 months	-0.016**	[-0.030,-0.001]	-0.008	[-0.021,0.005]
>6 months	-0.010*	[-0.021,0.000]	-0.008	[-0.018,0.002]
Delivery mode				
Normal	0.034	[-0.089,0.157]	0.027	[-0.077,0.132]
Assisted	0.039	[-0.085,0.163]	0.030	[-0.075,0.135]
Planned c-section	0.033	[-0.090,0.157]	0.033	[-0.072,0.138]
Emergency c-section	0.041	[-0.083,0.164]	0.04	[-0.064,0.145]
Alcohol in pregnancy				
Never	-	-	-	-
Light	-0.003	[-0.011,0.006]	-0.003	[-0.011,0.004]
Moderate	-0.024	[-0.040,0.008]	-0.024	[-0.039,0.010]
Heavy	-0.013	[-0.038,0.012]	-0.015	[-0.037,0.007]
Smoking in pregnancy				
Never	-	-	-	-
Gave up	-0.001	[-0.012,0.010]	0.004	[-0.006,0.013]
Kept smoking	0.004	[-0.006,0.014]	0.003	[-0.006,0.012]
Mother's BMI				
Underweight	-	-	-	-
Normal	-0.001	[-0.018,0.016]	0.007	[-0.008,0.022]

	RE (unweighted)		MLE (unweighted)	
	Coeff	**95% CI**	**Coeff**	**95% CI**
Overweight	-0.001	[-0.019,0.017]	0.006	[-0.010,0.022]
Obese	0.001	[-0.017,0.020]	0.012	[-0.005,0.028]
Mother's health				
Good or excellent health	-	-	-	-
Fair or poor health	0.020***	[0.010,0.029]	0.022***	[0.014,0.031]
Mother's mental health				
No or low distress	-	-	-	-
Medium distress	0.003	[-0.004,0.009]	0.002	[-0.004,0.008]
High distress	0.018**	[0.002,0.034]	0.017**	[0.003,0.031]
Smoking around child	-0.001	[-0.013,0.010]	0.004	[-0.006,0.014]
Damp	0.004	[-0.006,0.015]	-0.001	[-0.010,0.009]
Fume exposure	0.003	[-0.004,0.011]	0.008**	[0.002,0.015]
Rural-Urban morphology				
Urban	-	-	-	-
Mixed Urban-Rural	0.001	[-0.011,0.013]	-0.003	[-0.014,0.007]
Rural	-0.002	[-0.013,0.009]	-0.002	[-0.012,0.008]
Hausman p-value	0.428			

* p<0.10, **p<0.05, ***p<0.01

5.4.2. Approach 2 - Interaction between family income and MCS survey

The alternative method to analysing the association between income and asthma-related health outcomes in children and simultaneously take into account the longitudinal nature and allows the survey design of the MCS data to be taken into account. It does this by including an interaction term between income and each survey, i.e. MCS 2, MCS 3, MCS 4 or MCS 5. Table 5.11 and Table 5.12 display the marginal effects of family income for the unadjusted and fully adjusted models[38]. In the unadjusted models for outcome ever asthma, at age 3 a 1% increase in income was associated with a significant decrease in the probability of ever asthma of 0.055

[38] Extended fully adjusted model results with all included variables are reported in Appendix D-2.

(p-value: 0.001) (Table 5.11). The marginal effect was slightly less at age 3 in the fully adjusted model (marginal effect: -0.05; p-value: 0.001). Likewise adjusted marginal effects at ages 5, 7 and 11 are smaller than in unadjusted models with marginal effects -0.045 (p-value: 0.001); -0.025 (p-value: 0.01); -0.009 (p-value: 0.03) respectively.

With respect to wheezing in the last 12 months, the marginal effect of income in the unadjusted model was -0.04 at age 3 (p-value: 0.001). In the fully adjusted model the marginal effect at age 3 decreased in magnitude and was -0.03 (p-value: 0.001). In adjusted models, the marginal effect at age 5 also remained statistically significant (marginal effect: -0.04; p-value: 0.001). However, at ages 7 and 11 the association of income with wheezing in the last 12 months was no longer statistically significant in the fully adjusted model, in contrast to the unadjusted models with marginal effects -0.03 (p-value: 0.01) and -0.01 (p-value: 0.06) for unadjusted models at age 7 and 11 and marginal effects -0.01 (p-value: 0.17) and -0.01 (p-value: 0.37) for corresponding adjusted models (Table 5.11 and 5.12). For asthma medication use, the marginal effect of income at age 5 is -0.014 (p-value: 0.01). In the fully adjusted model, the marginal effect remains identical at age 5 (p-value: 0.01; 95% CI: -0.02,-0.004). Results from unadjusted and adjusted models at age 7 and 11 are also similar – marginal effects of income in fully adjusted models at ages 7 and 11 for asthma medication use are also statistically significant and are -0.01 (p-value:0.08) and -0.01 (p-value:0.03) respectively.

Table 5.11: Unadjusted marginal effects

	Ever asthma		**Wheezing last 12 months**		**Asthma medication use**	
	Marginal effect	**95% Conf Interval**	**Marginal effect**	**95% Conf Interval**	**Marginal effect**	**95% Conf Interval**
Age 3	-0.055***	[-0.070, -0.041]	-0.038***	[-0.053,-0.022]	-	-
Age 5	-0.062***	[-0.079,-0.045]	-0.041***	[-0.057,-0.025]	-0.014***	[-0.023,-0.006]
Age 7	-0.060***	[-0.077,-0.043]	-0.025***	[-0.040,-0.009]	-0.010**	[-0.020,-0.001]
Age 11	-0.053***	[-0.073,-0.033]	-0.014*	[-0.030,0.001]	-0.007**	[-0.018,-0.004]

* p<0.10, **p<0.05, ***p<0.01

Table 5.12: Fully adjusted marginal effects

	Ever asthma		Wheezing last 12 months		Asthma medication use	
	Marginal effect	**95% Conf Interval**	**Marginal effect**	**95% Conf Interval**	**Marginal effect**	**95% Conf Interval**
Age 3	-0.050***	[-0.073, -0.027]	-0.029***	[-0.050, -0.007]	-	-
Age 5	-0.045***	[-0.069, -0.021]	-0.039***	[-0.063, -0.016]	-0.014***	[-0.024,-0.004]
Age 7	-0.025***	[-0.043,-0.007]	-0.012	[0.030, 0.005]	-0.006**	[-0.012,-0.001]
Age 11	-0.009**	[-0.016,-0.001]	-0.002	[0.007, 0.003]	-0.006**	[-0.011,-0.001]

* p<0.10, **p<0.05, ***p<0.01

Determinants, other than income, that significantly increased the probability of ever asthma were family history of asthma, with marginal effects at ages 3, 5, 7 and 11 of 0.12 (p-value: 0.01), 0.10 (p-value: 0.001;) 0.07 (p-value: 0.01), and 0.03(p-value: 0.01), respectively (Appendix D-2). Corresponding figures at ages 3, 5, 7 and 11 for child sex were 0.05, 0.05, 0.03 and 0.01 (p-value:0.001 at all ages) respectively; for low birthweight 0.05 (p-value: 0.01), 0.05 (p-value: 0.01), 0.03 (p-value:0.03) and 0.01 (p-value: 0.01) and very low birthweight 0.15 (p-value: 0.001), 0.15 (p-value: 0.01), 0.10 (p-value: 0.02), 0.04 (p-value:0.003). For ever asthma, marginal effects for mother's general health, i.e. fair or poor health compared to good health at age 5, 7 and 11 respectively are 0.03 (p-value: 0.05), 0.03 (p-value: 0.02) and 0.01 (p-value: 0.08). Age 3 results for mother's general health were not statistically significant. Determinants that significantly reduce the probability of ever asthma are breastfeeding for more than 6 months at age 3, 5,7 and 11 with marginal effects -0.04 (p-value: 0.002), -0.03 (p-value: 0.002), -0.02 (p-value: 0.01) and -0.01 (p-value: 0.002) respectively (Appendix D-2).

Determinants that significantly increase the probability of wheezing in the last 12 months are family history of asthma at ages 3, 5, 7 and 11 with respective marginal effects 0.10 , 0.10, 0.05, and 0.02 (p-value: 0.001 for all ages). Child sex 0.06, 0.06, 0.03, and 0.01 for ages 3 to 11

respectively (p-value:0.001 for all ages); low birthweight, 0.04 (p-value: 0.03), 0.04 (p-value:0.02), 0.02 (p-value: 0.07); and 0.01 (p-value: 0.03) respectively with similar results for very low birthweight. Living in council rented homes also increased the probability of wheezing in the last 12 months compared to living in an owned or mortgaged home, marginal effects were 0.18 (p-value: 0.001); 0.20 (p-value: 0.001), 0.08 (p-value: 0.02) and 0.03 (p-value: 0.001) respectively; for privately rented homes corresponding figures were 0.15 (p-value: 0.001) 0.17 (p-value: 0.002), 0.06 (p-value: 0.02) and 0.02 (p-value: 0.001). Mother's mental health at age 3 also increased the probability of wheezing in the last 12 months by 0.03 (p-value: 0.01) for medium distress levels and 0.10 (p-value: 0.004) for high distress levels compared to low or no distress (Appendix D-2).

Determinants that significantly increase the probability of asthma medication use include family history of asthma at ages 5, 7 and 11 with marginal effects 0.03), 0.02, and 0.01 respectively (p-value: 0.001 for all ages). Marginal effects for child sex respectively were 0.01(p-value: 0.001) , 0.01 (p-value: 0.002), 0.01 (p-value: 0.001); low birthweight 0.02 (p-value: 0.03), 0.01 (p-value:0.06) and 0.01 (p-value:0.03); mother's general health (fair or poor health) 0.02 (p-value: 0.01), 0.01 (p-value: 0.02) and 0.01 (p-value: 0.06) respectively (Appendix D-2).

5.5. Discussion

5.5.1. Summary of results

In Approach 1, for outcome ever asthma, the FE model was preferable to the RE model based on the outcome of the Hausman test meaning the RE assumption of orthogonality does not hold. In the fully adjusted FE model, only in the unweighted model does income have a statistically significant association with ever asthma but this association is weak. In terms of

other determinants, the only time-variant factor that is associated with ever asthma is mother's general health, with children whose mothers have fair or poor health more likely to report ever asthma compared to mothers with good or excellent health, but this association is also weak.

In fully adjusted models for wheezing in the last 12 months, the FE estimate is preferable to the RE estimator. However, in both the weighted and unweighted FE models, income is not a statistically significant determinant of wheezing in the last 12 months. Child age is the only determinant that statistically offers an explanation to variation in wheezing in the last 12 months, with children who are older at the time of survey being less likely to have wheezing in the last 12 months. For asthma medication use, RE provides a more efficient estimator than the FE. In fully adjusted (unweighted) RE and (weighted) MLE models, but the association between income is only weakly statistically significant. However, unlike the ever asthma and wheezing in the last 12 month models, risk factors that one would expect to have a significant influence on child health outcomes, namely mother's general health, mother's mental health and breastfeeding duration are highlighted as being statistically significant risk factors in the RE model for outcome asthma medication use. However, the lack of findings with the other asthma-related outcomes signify a lack of a causal relationship rather than dismissing the importance of these factors at different points in childhood.

The results using panel data methods can be used to infer causality if the conditions of the models are fulfilled. However using panel data methods, the effect of income is only weakly associated across all outcomes, providing very little evidence to conclude that income itself is the underlying cause of ever asthma, wheezing in the last 12 months and asthma medication use. However, the effect of other risk factors is highlighted, some of which could potentially be targeted by policy intervention, specifically, low levels of education in mothers and poor general and mental health in mothers.

For Approach 2, adjusted results for ever asthma indicate that the effect of income significantly decreases from age 3 to age 11. For wheezing in the last 12 months, only results at age 5 and age 11 were statistically significant and for asthma medication use, the association decreased from age 5 to age 7 and remained the same at age 11. Across all outcomes and ages, mother's general health and mental health were found to be strongly associated with asthma-related outcomes.

When comparing the two approaches – there are both similarities and differences between the results. For outcome ever asthma, the FE for Approach 1 shows a weak association with income, which is similar in magnitude to the marginal effect at age 11 in Approach 2, although the latter is strongly significant. Whilst wheezing in the last 12 months is not significantly associated with family income in any of the fully adjusted models in Approach 1, it is in Approach 2 with a highly significant association between income and wheezing in the last 12 months at ages 3 and 5 but not after. This difference could be explained by the fact that if income is a not a determinant of wheezing in late childhood, then one might expect the overall effect of income as expressed in Approach 1, to be very low or perhaps not statistically significant, as was seen in the results. For asthma medication use, the estimate for the income effect in Approach 1 (RE model) is similar to the associations in Approach 2 at ages 5, 7 and 11. To the best of my knowledge, the only paper that looks at the relationship between income and asthma using panel data is Violato et al (2009). They also used a linear probability FE model to analyse the association between family income and child respiratory health using MCS data in children from 9 months to 5 years. The coefficient of income estimated in the Violato et al (2009) study was -0.012 (p-value: 0.13) and, although the results from my analysis used a different age range (ages 3 to 11) as well a different set of determinants, the results are not far

off from the adjusted unweighted FE model for outcome ever asthma, with a coefficient of -0.01 (p-value:0.08) in the unweighted model.

5.5.2. Strengths and limitations

In this chapter I exploited the longitudinal nature of the MCS, using panel data methods (Approach 1) and an ad hoc approach using interaction terms between time measured by MCS survey and income (Approach 2) to assess whether the association between family income and asthma-related outcomes exists over time. This approach offered a dynamic analysis to add value to the cross-sectional analysis that was performed in Chapter 3 that is most often used across the literature investigating the association between income and child health outcomes, but not applied often to specific child health outcomes.

The main strength of this analysis is that it makes full use of the rich information at individual and family level that the MCS measured repeatedly, over time. This allowed me to investigate the relationship between family income child asthma-related outcomes, fully exploiting the longitudinal dimension of the dataset. Panel data methods take into account variation between individuals (FE) and within individuals (RE) and can infer causality. The results of this chapter offer new insights for the economic research of family income and asthma-related outcomes as it uses a follow-up period of 11 years and explores different econometric possibilities.

Limitations include the use of subjective measures of asthma-related outcomes which may not be accurate and present recall biases. There were also a limited number of time varying factors that could be included in RE and MLE estimation which could result in biased results as it does account for omitted variables. FE estimations allow for omitted time-invariant variables by

controlling out, rather than explicitly modelling such variables which may produce overly simplified models which could lead to misleading findings (Hill et al, 2020).

5.6. Conclusion

The longitudinal methods used in this chapter exploit the panel nature of the MCS data,. Findings show that the causal effect of income is weak in the case of outcome ever asthma (FE) and asthma medication use (RE) (Approach 1). This may be due to the limited variability in family income across the time periods included in the analysis.

6. Income-related inequality in child asthma-related outcomes

6.1. Introduction

Across countries, differences in health outcomes in children within the same population have been found to be partly determined by underlying socioeconomic differences (Case et al., 2002; Currie and Stabile, 2003; Propper et al., 2006). These differences are found not only in the case of general health, but also in specific health conditions such as asthma-related outcomes (Violato et al., 2009). Chapters 3, 4 and 5 of this thesis applied different methodologies and altogether concluded that there is a positive and statistically significant association between low family income and adverse asthma-related outcomes in children, which supports what has been found in previous literature (Chapter 1).

However, in order for policy makers to address differences in health outcomes with adequately targeted policies, it is necessary to measure how ill-health is distributed across different socioeconomic groups within the population. More concisely – measuring the extent of socioeconomic-related inequality in the health of a population. Establishing a measure of inequality could then allow for determinants of inequality to be calculated. Insight into factors driving inequality could lend a hand in better highlighting areas where changes in policy could be effective (Costa Font et al., 2011; van Doorslaer and Koolman 2014).

There has been an increased interest in income-related inequality in health in recent years, especially in health policy research. This is because the disparity in health in low-income groups compared to health in high income groups is concerning in many countries, including the UK, with the poorest in society having worse health than their wealthier counterparts (O'Donnell et

al., 2008; McCartney et al., 2019). Although nowhere near the extremes of inequalities seen in low and middle-income countries, income inequality in the UK is relatively substantial when compared to other European countries, with a Gini coefficient of 0.35 and the 4th most unequal in Europe (Deaton, 2003; Marmot, 2010; RCPCH, 2020; Chancel, 2019).

The conclusion that equal societies are healthier is widely acknowledged and has been incorporated into national health strategies. The mandates of the devolved governments of the UK, to public health bodies such as NHS England[39], include the improvement of local and national outcomes by specifically addressing health inequalities (Department of Health and Social Care, 2019).

For children, in particular, addressing inequality in health is of utmost importance as it is the early experiences that lay a foundation for the rest of their lives. Good foundations in a child's early years can greatly reduce inequalities in health across their life course (Marmot, 2020). In the 2020 State of Child Health report (for England), it was found that across all health indicators, including asthma-related outcomes, children from deprived backgrounds had much worse health and wellbeing than their wealthier counterparts; the report showed that for children aged 1-4, there were 400 emergency asthma admissions for every 100,000 children in the most deprived quintile, compared to 200 cases for every 100,000 in the least deprived quintile - almost twice as many cases in the most deprived group (RCPCH, 2020). For children aged 5-9, these figures were slightly lower, 350 emergency asthma admissions for every 100,000 children in the most deprived quintile compared to 150 admissions in the least deprived quintile

[39] Respective bodies in Northern Ireland, Scotland and Wales are the Public Health Agency, Public Health Scotland and Public Health Wales

(RCPCH, 2020). These figures were comparable across the whole of the UK, further emphasising the need to tackle the underlying causes of inequality in health.

This chapter outlines different measures of income-related inequality in health . I then use MCS data to estimate indices to quantify the extent of income-related inequality in asthma-related outcomes in UK children, and perform a decomposition analysis to highlight factors driving the observed inequality.

6.2. Measuring income-related inequality in health

Several indices have been used in the economic literature to measure inequality in health, the choice of which depends on the measurement qualities possessed by the index (Costa-Font and Hernández-Quevedo, 2012; van Doorslaer and Koolman, 2004; Walsh and Cullinan, 2015). The most commonly used measures of inequality in health, as seen in economic literature, are:

- the Range;
- the Lorenz curve and Gini coefficient;
- the pseudo Lorenz curve and pseudo Gini coefficient;
- the Index of Dissimilarity;
- the Slope Index of Inequality and Relative Index of Inequality;
- the Concentration Index;

These each come with advantages and limitations. The Range was once the most commonly used measures of inequality in both economic and health inequality literature (Wagstaff et al., 1991; Heshmati, 2006). It measures inequality by comparing the experiences of the top and bottom socioeconomic groups in a given population, by taking the difference between these groups as expressed in equation (6.1), where experiences can be defined as quantitative

measures of health outcomes. The Range is often further extended where inequality is represented as a ratio of one extreme over the other as in equation (6.2), i.e. the average health outcome of the top socioeconomic group over the average health outcome of the lowest socioeconomic group. The Range in its two forms can be expressed as:

$$R = y_{max} - y_{min} \quad (6.1)$$

$$R_r = \frac{y_{max}}{y_{min}} \quad (6.2)$$

where R stands for range; y_{max} and y_{min} represent the maximum and minimum health outcomes of the top and the lowest population groups, respectively; and R_r is the range expressed as a ratio. The advantage of the Range is that it considers health outcomes of both ends of the socioeconomic distribution of the population – the lowest and highest socioeconomic groups. It works particularly well as a measure of inequality in small populations where there is a small number of socioeconomic groups and these minimum and maximum values of the outcome of interest can easily be determined (Wagstaff et al., 1991; Heshmati, 2006). The main disadvantage of the Range is that it overlooks changes in inequality that occur in the intermediate socioeconomic groups, particularly in larger populations with differential distribution in health, because it does not account for the whole spectrum of income distribution in the population under analysis. In addition, the range is not standardised for population size as it does not account for the size of its top and bottom socioeconomic groups (Cowell, 2011).

Originally, the Lorenz curve was used to graphically represent the income distribution in a population of interest depicted on a Cartesian plane, with the defined population on the horizontal axis and the cumulative percentage of income or wealth on the vertical axis (Cowell 2011). However, Lorenz curves can also be used to depict distributions other than income, including health outcomes in a population. In this instance, the Lorenz curve plots the

cumulative percentage of health using individual-level data, starting with the sickest individual, on the vertical axis, against the cumulative population ranked by health, on the horizontal axis (Figure 6.1). The Lorenz curve coincides with the 45-degree line running from the origin if the health variable is equally distributed in the population, and the further the curve lies from the 45-degree line, in any direction, the greater the degree of inequality (Wagstaff et al., 1991; O'Donnell et al., 2008). Inequality is then quantified by the Gini Coefficient (G), which is defined as two times the area between the Lorenz curve and the 45-degree line and is calculated as:

$$G = \frac{\sum_{i=1}^{n-1} |p_i - q_i|}{\sum_{i=1}^{n-1} p_i} \quad (6.3)$$

where p_i represents the proportion of individuals ranked by health level and q_i represents the cumulative total proportion of health of these individuals (Wagstaff et al., 1991; O'Donnell et al., 2008; Cowell, 2011). The biggest advantage of the Lorenz curve is that unlike the Range, it considers the entire population. However, it does not incorporate the socioeconomic dimension of inequality in health (Wagstaff et al., 1991). This is a major disadvantage, especially for the purposes of this research, as I am interested in not only inequality in health, but also the way the health is distributed in relation to income level.

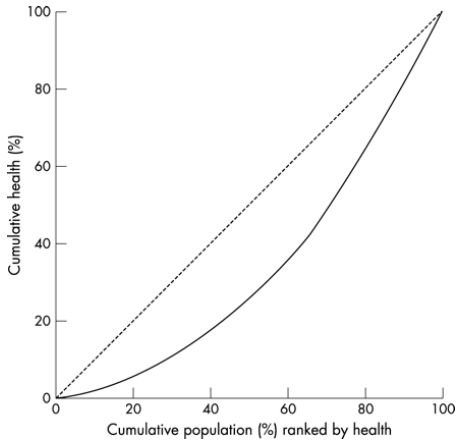


Figure 6.1: The Lorenz curve for health

Another measure of inequality is the Index of Dissimilarity which takes into account, for each socioeconomic group in a population, the absolute value of the difference between the group's share of health and how much of the population they make up, where a greater difference indicates a greater level of inequality (Wagstaff et al.,1991; Cowell, 2011). The Index of Dissimilarity is calculated as:

$$ID = \frac{1}{2} \sum_j |S_{jh} - S_{jp}| \quad (6.4)$$

where S_{jh} is the j^{th} group's share of the population health, h and S_{jp} is the j^{th} group's share of population, p. An advantage of the Index of Dissimilarity is that it considers the entire population and health ranking but like the Lorenz curve, it does not consider socioeconomic ranking in relation to health. The following measures of inequality attempt to address this limitation (Wagstaff et al., 1991).

The Slope Index and related Relative Index of Inequality are two measures that do reflect the socioeconomic dimension of inequality in health. To obtain the Slope Index and Relative Index of Inequality, the mean health status of each socioeconomic class in the target population is calculated and then ranked by the socioeconomic class (Wagstaff et al., 1991). The Slope Index of Inequality can be defined as "*the slope of the regression line that shows the relationship between a class's health status and its relative rank in the socioeconomic distribution*" (Wagstaff et al., 1991). The interpretation of the Slope Index is the absolute effect, on health, of moving from the lowest to the highest socioeconomic group. Generally, the Slope Index of Inequality is sensitive to the mean health of the population under study however, when it is divided by mean health, any changes in the mean health in the population will then leave the index unaffected (Wagstaff et al., 1991). This subsequent measure, i.e. the Slope Index divided by mean health, is known as the Relative Index of Inequality (Wagstaff et al., 1991; O'Donnell et al., 2008). One drawback

of the Slope Index and Relative Index of Inequality is that they are calculated using grouped socio-economic data rather than individual-level data, the latter being more reflective of the entire population.

The final measure of inequality is the Concentration Index (CI) and associated concentration curve. The CI ranks a population by socioeconomic status, beginning with the most disadvantaged using individual-level data. This immediately fulfills the shortcomings of the measures discussed earlier, and making it a suitable measure of income- related health inequality.

6.3. Concentration curves and the Concentration Index

Concentration curves have frequently been used in the literature to graphically depict income-related health inequality. These curves plot the cumulative percentage of the health variable, on the vertical axis, against the cumulative percentage of the population sample, ranked usually by income or another standard of living measure, on the horizontal axis (Wagstaff et al., 1991). Differences in the proportion of ill-health at different points along the income distribution of a population can be used to investigate the degree of inequality in health (Wagstaff et al., 1991; Kakwani et al., 1997; O'Donnell et al., 2008). Figure 6.2 represents a concentration curve, which shows the relationship between income and ill-health. The 45-degree line is the line of health equality, which indicates a situation in which health is equally distributed across the entire population. Deviations from the line of equality represent a degree of health inequality (Wagstaff et al., 1991; Kakwani et al., 1997; O'Donnell et al., 2008). A concentration curve that lies above the line of equality indicates that ill-health is concentrated amongst those with low income and one falling below the 45-degree line is indicative of ill-health being concentrated amongst people with high income. Figure 6.2 is an example that depicts the former, i.e. that ill-

heath as measured by 'ever asthma' in 3 year olds is disproportionately concentrated among children from low-income families.

A concentration curve however cannot be used as a standalone tool as it cannot give a measurable magnitude of inequality. However this is overcome by using it together with the related CI, which represents a quantifiable measure of inequality in health and also considers a socioeconomic dimension in its calculation, which is ideal for the objectives of this chapter. The value of the CI can easily be calculated numerically as twice the area between the concentration curve and the 45-degree line of equality (Wagstaff et al., 1991; Kakwani et al., 1997; O'Donnell et al., 2008).

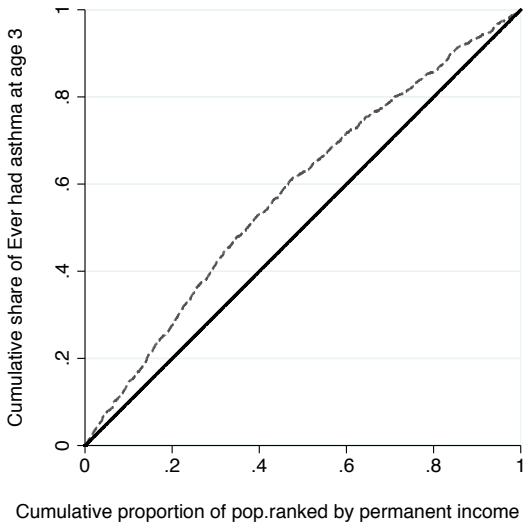


Figure 6.2: The Concentration Curve (source: Millennium Cohort Study)

The CI measures the degree of socioeconomic related inequality in a health variable and is most commonly expressed as:

$$CI = \frac{2}{N\mu} \sum_{i=1}^{N} h_i R_i - 1 \quad (6.5)$$

where h_i is the health outcome for individual *i*, μ is its mean and R_i is the fractional rank of individual *i* in the income distribution, and is equivalent to $R_i = \frac{i}{N}$ where *i*=1 for the poorest and *i*=*N* for the richest (Wagstaff et al., 1991; Kakwani et al., 1997; O'Donnell et al., 2008). To simplify the computation, the CI can also be expressed as the covariance between individual health h_i and an individual's income rank R_i scaled by the mean health of the population, μ:

$$CI = \frac{2}{\mu} cov(h_i, R_i) \quad (6.6)$$

6.3.1. Interpreting the Concentration Index

The value of the CI is bounded between values -1 and +1 with value of zero indicating that ill-health is distributed evenly across the income distribution in a given population (Wagstaff et al.,1991; O'Donnell et al., 2008). A negative CI value indicates that ill-health is disproportionately concentrated amongst those with low income. Conversely, a positive CI value is indicative of ill-health being disproportionately concentrated amongst people with high income (Wagstaff et al., 1991; O'Donnell et al., 2008). The higher the absolute value of the CI, the more extreme the inequality is, in either direction (Erreygers, 2009).

6.3.2. Shortcomings of the Concentration Index

Firstly, when the CI is used as a measure of inequality for a health variable that has a finite positive upper value and positive lower value, including binary variables, it has been observed that the bounds of the index then depend on the mean of the health variable and will not necessarily fall between -1 and 1 (Wagstaff, 2005; Erreygers, 2009). The bounds of the CI turn out to be much wider where the mean health for a population is low (closer to 0) compared to populations with a high mean health (closer to 1). The bounds shrink to $\mu - 1$ (lower bound)

and $1 - \mu$ (upper bound) where μ is the mean of the health variable (Wagstaff, 2005; Erreygers 2009).

Secondly, the perspective of the health variable may create distortion in the CI. To elaborate, a health variable can be expressed either in terms of health or ill-health which are essentially mirrors of one another. One would expect the CI of two populations being compared, whether measured by ill-health or (good) health, to give the same degree of inequality – the indices should be mirror images (i.e. identical absolute values) but with the opposite sign. However, studies have shown that health ranking within the same population can vary depending on whether health was measured as good-health or ill-health, and this distorts the comparison of inequality (Kjellsson and Gerdtham, 2013). The ideal situation would be for the CIs, regardless of whether they are measured by good-health or ill-health, to be mirror images of one another (same magnitude, opposite sign) (Erreygers, 2009).

The third shortcoming is related to the properties of the measure of health. If the health measure is ordinal or cardinal, the CI value will change if there are positive monotonic transformations or positive linear transformations of the health measure, respectively. Where the health variable is measured on a ratio-scale, a positive proportional transformation of it will leave the CI unchanged, which means that one would be able to compare differing ranked health states and that the point of zero health is fixed, unambiguously. This shortcoming is known as the "Cardinality issue" (Wagstaff, 2005; Erregers, 2009).

6.3.3. Solutions to the shortcomings of the Concentration Index

In light of the shortcomings outlined, there are adjustments that can be made to correct the CI to make it more suitable to measuring income inequality in health with the data used in this thesis.

6.3.3.1. The Wagstaff normalisation

The Wagstaff normalisation is specifically aimed at addressing the bounds issue of the CI that occurs specifically when the health outcome is binary (Wagstaff, 2005). As specified before, when a variable whose distribution is being analysed is unbounded, the CI falls between -1 and +1, but this is not the case when the variable is binary. In my research the three binary health outcomes used are: whether a child is reported to having asthma or not (ever asthma); wheezing in the last 12 months or not; using asthma medication or not. The correction is a simple multiplication of the CI by a normalisation factor. The result is the Wagstaff normalised Concentration Index (hereon referred to as the Wagstaff Index (WI)). The normalisation can be represented as:

$$WI = \frac{CI}{1-\mu} \quad (6.7)$$

The WI shown in equation (6.7) is calculated by dividing the CI by 1- μ (where $\frac{1}{1-\mu}$ is the normalisation factor), and μ is the mean of the health outcome (Wagstaff, 2005; Erreygers, 2009; Wagstaff, 2009; Walsh and Cullinan, 2015). This normalisation ensures that the bounds of the CI are not distorted by changes in mean health.

6.3.3.2. Generalised Concentration Index

A normalisation developed to deal with differences resulting in CI depending on whether health or ill-health is used, is the Generalised Concentration Index (GCI) first developed by Wagstaff et al (1991). It is calculated by multiplying the CI either by the mean of the good-health outcome (μ_h) or mean ill-health outcome (μ_{ih}):

$$GCI_h = \mu_h CI_h \quad (6.8)$$

$$GCI_{ih} = \mu_{ih} CI_{ih} \quad (6.9)$$

In the equations (6.8) and (6.9) above, GCI represents the Generalised Concentration Index of the good health h, or ill-health measure ih and, CI is the Concentration Index. It can be seen that:

$$CI_{ih} = -\frac{\mu_h}{\mu_{ih}} CI_h \quad (6.10)$$

Therefore GCI_h = -GCI_{ih}, which fulfils the mirror condition.

6.3.3.3. Erreygers' correction

Another corrected form of the CI was developed by Erreygers (2009). Erreygers aimed to address all the shortcomings of the CI, the issues of boundedness, the mirror condition and the issue of cardinality. Erreygers observed that if four key requirements are met, then all the shortcomings of the CI would be addressed (Erreygers, 2009). These four requirements are:

- Transfer – if there is a mean-preserving change in the health distribution, where the health of an individual *i* decreases and the health of individual *j* increases by the same amount, the value of the index will increase if individual *i* is richer than *j*, and decrease

if *i* is poorer than *j*. In other words, if the change in the distribution health favours the better-off individual (in socioeconomic terms) this change is defined as being "pro-rich", if the change in the distribution of health favours the worse-off the change is defined as being "pro-poor";

- Level independence – if the health level of all persons in a given health distribution changes by the same absolute amount, the value of the index remains unchanged (Erreygers, 2009; Erreygers and Van Ourti, 2011; Kjellsson and Gerdtham, 2013);
- Cardinal invariance – a positive linear transformation of the health variable does not have an effect on the value of the index (Erreygers 2009; Erreygers and Van Ourti, 2011; Kjellsson and Gerdtham 2013); and
- The mirror condition – for any health distribution, the health index (calculated using a positive health outcome) and associated ill-health index (calculated using morbidity) are equal in absolute value but have opposite signs (Erreygers, 2009; Kjellsson and Gerdtham 2013).

The correction that takes into account the four requirements results in the Erreygers' Index (EI), and is represented by:

$$EI = 4\frac{\mu}{h_{max}-h_{min}}\ CI \quad (6.11)$$

Where h_{max} is the upper bound and h_{min} the lower bound of the health variable h, with mean μ. The term $h_{max} - h_{min}$ is equal to 1, in the case of a dichotomous health variable. The EI fulfils all of the above requirements of transfer, level dependence, cardinal invariance and the mirror condition. The EI has therefore been selected for use in my analysis for the following reasons:

- it considers both the health and socioeconomic dimension in the study population;
- it takes into account the whole population and uses individual-level data for both health and socioeconomic variables without omitting intermediate socioeconomic groups in the middle of the population distribution. Thus it gives an accurate estimate of the level of income-related inequality in the entire population;
- Has a graphical representation in concentration curves;
- it is sensitive to changes in the distribution of the population. For example, a transfer in income between two households in which the income ranking is changed is reflected by changes in the EI (Wagstaff et al., 1991; O'Donnell et al., 2008);
- it is suitable to use when the health outcome is binary, with minimum bound of -1 and maximum bound of +1;
- when compared to other indices (e.g. the CI, WI, GCI), the EI satisfies all of the four abovementioned conditions whereas the CI only satisfies the transfer and cardinal invariance conditions; the WI satisfies all conditions except the level independence condition; and the GCI satisfies all except the cardinal invariance condition.

6.4. Decomposition of the Erreygers' Index

The CI and associated indices (EI, GCI, and WI) can be decomposed to identify contributing factors (Kakwani et al., 1994; Wagstaff et al., 2003; O'Donnell et al., 2008). In terms of the CI, any additive linear regression model of a health outcome (h) is estimated as:

$$h = \alpha + \sum_k \beta_k x_k + \varepsilon \quad (6.12)$$

The CI can therefore be expressed as:

$$CI = \alpha + \sum_k \left(\frac{\beta_k \bar{x}_k}{\mu}\right) CI_k + \frac{GCI_\epsilon}{\mu} \quad (6.13)$$

where μ is the mean of the health outcome y, x_k represents the regressors which explain the observed inequality, β_k is the regression coefficient of determinant x_k; $\bar{x}_k$ is the mean of x_k; CI_k is the individual CI for x_k and GCI_ϵ is the GCI for the error term (ϵ). Equation (6.13) can be modified to decompose the EI (Van de Poel et al., 2012; Hong and Lee, 2019):

$$EI = 4\sum_k (\beta_k \bar{x}_k) CI_k + GCI_\epsilon \quad (6.14)$$

Equation (6.14) indicates that the EI can be thought of as being made up of two components. The first being the deterministic component which is equal to a weighted sum of the all the individual CIs of all k regressors where the weight of x_k is the elasticity of health outcome y with respect to x_k, that is β_k. The second term, GCI_ϵ, is the generalized concentration index of the error and reflects the socioeconomic inequality that is left unexplained by the model, i.e. it captures the residual component (O'Donnell et al., 2008; Walsh and Cullinan, 2015). Equation (6.14) shows that total inequality can be partitioned into inequality that is associated with each of the regressors (Van Doorslaer and Koolman, 2004; Wagstaff et al., 2003; Hernández-Quevedo et al., 2006; Hong and Lee, 2019). The β coefficients in equation (6.14) are derived from the linear regression of the health outcome (y) for each (k).

The contribution of each individual determinant factor x_k is decomposed into two components 1) its impact on health as measured by the elasticity of the health variable with respect to each factor (β_k) and, 2) the degree of unequal distribution as measured by CI_k (Wagstaff et al, 2003).

To the best of my knowledge there have been no studies that have specifically measured income-related health inequality using the CI (or EI and WI) for asthma-related outcomes in

UK children. Consequently, decomposition analysis to identify contributing factors to income-related inequality in child asthma-related outcomes is also novel.

6.5. Data

For the analysis, the main determinant factor is family income. Permanent income is used in calculating the EI and associated indices to account for long-run income. For analyses at age 3, permanent income takes into account family income at age 3; for the age 5 analyses, permanent income at age 5 considers cumulative family income at ages 3 and 5 and so on (Chapter 2, Section 2.9). Three asthma-related outcomes are operationalised as health outcomes for the analysis. These are: ever asthma, wheezing in the last 12 months and asthma medication use. These outcomes were derived from the responses to MCS questions asked to the mothers of the cohort children (Chapter 2, Section 2.8).

Potential determinant factors included in the decomposition analysis are similar to those used in previous chapters with four groups of determinant factors being included: a priori factors which include child ethnicity, a history of asthma in the family, birthweight, child age at time of survey and number of siblings at the time of the first MCS survey at 9 months; measures of socioeconomic status other than family income housing tenure, mother's highest level of education and mother's age at time of birth of the cohort child; mother and child health-related variables, namely maternal smoking and alcohol consumption during pregnancy, mother's BMI at birth of child, mother's general health and mother's mental health and; household and environmental factors, namely damp and condensation in the home, child exposure to cigarette smoke in the home, exposure to fumes from heating and rural-urban morphology (determinants are described in (Chapter 2, Section 2.10.).

6.6. Analysis

With respect to the methods and data described in Sections 6.4 and 6.5, the analysis section of chapter consisted of two steps:

1. **Income-related inequality was estimated using** the EI for the base case (CIs and WIs estimated for comparison) for each health outcome: ever asthma (ages 3, 5, 7 and 11), wheezing the last 12 months (ages 3, 5, 7 and 11) and asthma medication use (ages 5, 7 and 11).
2. **Statistically significant EIs were then decomposed** into components– individual elasticity β_k, the individual concentration index CI_k, the contribution of each factor to the overall EI (product of β_k and CI_k) and the percentage contribution, which was calculated as the individual contribution divided by the overall EI. This was performed for every determinant included in the model. Modified user-written Stata syntax was used to perform the various index estimations and decomposition analysis (O'Donnell et al., 2016).

6.7. Results

6.7.1. Erreygers' Indices

In Table 6.2, results for the outcome ever asthma show that EIs are -0.08 (p-value:0.001) for children at ages 3, 5 and 7, and -0.06 (p-value:0.17) at age 11, indicating significant income inequalities across all ages under analysis, except for age 11.

Table 6.2: Erreygers', Concentration & Wagstaff Index: Ever asthma

	Age 3	**Age 5**	**Age 7**	**Age 11**
Erreygers' Index	-0.076***	-0.082***	-0.077***	-0.062
Concentration Index	-0.161***	-0.143***	-0.123***	-0.088*
Wagstaff Index	-0.183 ***	-0.167***	-0.140***	-0.150
Sample size	9897	9979	9882	9892

p< 0.10 *; p<0.050 **; p<0.001 ***

The corresponding CI and WI are also negative and statistically significant indicating the inequality in the same direction as the EI, ever asthma is distributed more heavily in low-income children. Concentration curves corresponding to the calculated EIs are depicted in Figure 6.3[40]. It can be noted that the curves lie above the 45 degree line at all ages. For the outcome wheezing in the last 12 months (Table 6.3), EIs are negative and significant across all ages. At ages 3 and 5 the EI is -0.06 (p-value <0.001) At age 7, the EI increases to -0.03 (p-value<0.001); and at age 11 to -0.02 (p-value:0.070). Corresponding CIs and WIs are also negative and statistically significant across all ages.

Table 6.3: Erreygers', Concentration and Wagstaff Index: Wheezing in the last 12 months

	Age 3	**Age 5**	**Age 7**	**Age 11**
Erreygers' Index	-0.056***	-0.058 ***	-0.030***	-0.016*
Concentration Index	-0.073***	-0.092***	-0.065***	-0.036**
Wagstaff Index	-0.090***	-0.109 ***	-0.074***	-0.040*
Sample size	10062	10018	9920	9920

p< 0.10 *; p< 0.050 **; p<0.001 ***

Concentration Curves corresponding to the EIs are displayed in Figure 6.4. The curves lie closer to the 45 degree line compared to the case of ever asthma, which indicates less income-related inequality in wheezing in the last 12 months. Table 6.4 reports EIs for the income-related

[40] Figure 6.3, 6.4 and 6.5 are at the end of this chapter, page 207, 208, 209

inequality in asthma medication use, for ages 5, 7 and 11. Similarly to the other two outcomes, EIs are also negative but statistically significant only for ages 5 (EI: -0.02; p-value<0.01) and 7 (EI: -0.01; p-value: 0.02). At age 11 it is still negative but not significant (EI: - 0.01; p-value: 0.002). These results once again indicate that there is income-related inequality in asthma medication use which is concentrated amongst children from low-income families. The change in magnitude of the EI indicates that income-related inequality in asthma medication use appears to have lessened between ages 5 and 7, although the EI is not statistically significant at age 11. CIs and WIs are negative and statistically significant at age 5 and 7.

Table 6.4: Erreygers', Concentration and Wagstaff Index: Asthma medication use

	Age 5	**Age 7**	**Age 11**
Erreygers' Index	-0.021***	-0.013**	-0.008
Concentration Index	-0.135***	-0.071***	-0.039
Wagstaff Index	-0.140***	-0.075**	-0.041
Sample size	10019	9903	9895

p< 0.10 *; p<0.050 **; p<0.001 ***

Concentration Curves for the EI for asthma medication use are presented in Figure 6.5. It can clearly be seen that the magnitude of the index decreases between ages 5 and 11 as the curve shifts closer to the 45 degree line whilst remaining above it, indicating income-related inequality, to a lesser extent compared to the other outcomes.

6.7.2. Decomposition analysis

The individual EI for each determinant variable/factor are shown in the first column of each results table. Positive EIs indicate that a factor is more highly concentrated or more prevalent amongst richer individuals and a negative EI indicates the contrary, i.e. that the determinant factor is more concentrated in poorer individuals. The second column shows the elasticity of the health outcome with respect to that factor (Elas). The larger the magnitude of the elasticity,

the more sensitive the health outcome is to changes in the factor. A negative elasticity indicates that the change in the health outcome and the change in the factor are in opposite directions. The third column shows the specific contribution (Cont) of each factor to the overall inequality and is given by the product of the individual fact or EI and elasticity of the health outcome with respect to that factor (Elas). The contribution to the overall EI may be driven by high elasticities or by high income-related inequality for that specific factor because the contribution is the product of elasticity and the EI for each factor. The direction of the effect of each factor contribution, depends on the sign of the overall EI. If the overall EI is positive, an individual factor with a positive contribution adds to income-related inequality. An individual factor with a negative contribution, will reduce the overall EI. Where the overall EI is negative, an individual factor with negative contribution sign adds to inequality and a factor with a positive contribution reduces the overall inequality in this case. The forth column represents the percentage contribution (% Cont) of each determinant factor. This is the individual contribution of each variable expressed as a percentage of the overall EI. A positive percentage contribution indicates that the variable contributes positively to the overall EI and has the same sign, whilst a negative percentage contribution indicates that the variable reduces the overall EI and its contribution has the opposite sign to the overall EI. For categorical variables, the overall % Cont of the variable is obtained by summing the % Cont of each category of that variable.

6.7.2.1. Ever asthma

For ever asthma at age 3 the overall EI is -0.08 (Table 6.5), as was seen previously. Family income, which has a negative sign for contribution, is a major contributor to overall income inequality in ever asthma, contributing 36%. The large elasticity of ever asthma with respect to income (i.e. -0.799) is responsible for its larger contribution, more so than inequality in income (0.035). Another major contributor to income-related inequality in ever asthma, is housing

which in aggregate[41] [42]contributes 37% to the EI. Results show that contribution is driven by the large negative EIs for each category, -0.61 for council housing, -0.42 for privately rented housing and -0.75 for other housing compared to owned or mortgaged homes, showing that the income-related inequality in housing is a main driver of inequality in ever asthma at age 3.

On aggregate, income-related inequality in breastfeeding duration contributes 14% to income-inequality and is driven mainly by individual EIs rather than elasticity. Income-related inequality in other mother and child health-related factors that contributed towards to the inequality in ever asthma are mother's mental health (6%), mother's general health (4%), smoking in pregnancy (4%) and alcohol consumption during pregnancy (3%) which again was driven by negative EIs for each category, rather than elasticity. Income-related inequality in factors that contributed negatively were mother's education (-16%) and mode of delivery (-2%), driven largely by positive EIs. The residual component is -0.007 and indicates that about 9% of the income-related health inequality is not explained by systematic variation in the regressors (Table 6.5).

Table 6.5: Decomposition of the EI: Ever asthma age 3

		EI	**Elas**	**Cont**	**% Cont**
Ln income		0.035	-0.799	-0.028	36.38%
Child age		0.001	0.547	0.001	-0.80%
Child sex (male)		0.010	0.080	0.001	-1.04%
Birthweight:					
	Normal	ref	ref	ref	ref
	Low	-0.132	0.008	-0.001	1.42%
	Very Low	-0.094	0.004	-0.0004	0.49%
Family history of asthma		-0.002	0.123	-0.0002	0.39%
Number of siblings					
	No siblings	ref	ref	ref	ref
	1 sibling	0.032	0.013	0.0004	-0.54%
	2 siblings	-0.157	0.002	-0.0003	0.33%

[41] Aggregate as calculated by the % contribution for each category of housing: 25.26% + 12.78% - 0.81% = 37%.

[42] The contribution of each determinant factor is a product of the income-related inequality in that factor and its elasticity with respect to the health outcome. Large negative individual EIs therefore signal a large degree of income-related inequality in that factor.

		EI	Elas	Cont	% Cont
	3 or more siblings	-0.405	0.006	-0.002	3.03%
Mother's education					
	No qualification	ref	ref	ref	ref
	NVQ level 1	-0.125	0.001	-0.0001	0.21%
	NVQ level 2	0.004	-0.008	-0.000	0.04%
	NVQ level 3	0.356	0.024	0.008	-11.05%
	NVQ level 4	0.600	0.004	0.002	-3.10%
	NVQ level 5	-0.408	0.0001	-0.000	0.02%
	Overseas qualification	-0.602	-0.002	0.001	-1.61%
Housing					
	Owned or mortgaged	ref	ref	ref	ref
	Council rented	-0.609	0.032	-0.019	25.26%
	Rented privately	-0.420	0.023	-0.010	12.78%
	Other housing	-0.750	-0.001	0.001	-0.81%
Mode of delivery					
	Normal vaginal	ref	ref	ref	ref
	Assisted	0.145	0.001	0.000	-0.25%
	Planned c-section	0.056	0.009	0.000	-0.65%
	Emergency c-section	0.124	0.006	0.001	-1.03%
	Other	-0.230	0.000	-0.000	0.06%
Breastfeeding duration					
	Never	ref	ref	ref	ref
	<2 months	-0.012	-0.008	0.000	-0.12%
	2-4 months	0.150	-0.013	-0.002	2.60%
	4-6 months	0.279	-0.009	-0.002	3.16%
	>6 months	0.159	-0.042	-0.007	8.78%
Mother's general health					
	Good or excellent	ref	ref	ref	ref
	Poor or fair	-0.265	0.011	-0.003	3.90%
Mother's mental health					
	Low distress	ref	ref	ref	ref
	Medium distress	-0.123	0.013	-0.002	2.12%
	High distress	-0.468	0.006	-0.003	3.65%
Alcohol consumption					
	None	ref	ref	ref	ref
	Light	0.188	-0.011	-0.002	2.74%
	Heavy	0.064	-0.001	0.000	0.05%
Smoking in pregnancy					
	Never smoked	ref	ref	ref	ref
	Gave up smoking	-0.051	0.004	-0.000	0.24%
	Kept smoking	-0.375	0.008	-0.003	3.84%
Mother's BMI at birth					
	Normal	ref	ref	ref	ref
	High	0.068	0.016	0.001	-1.42%
	Low	-0.019	0.011	-0.000	0.28%
Smoking around child (yes)		-0.429	-0.002	0.001	-1.20%
Damp in the home (yes)		-0.267	0.000	0.000	-0.07%
Fume exposure (yes)		-0.046	0.015	-0.001	0.93%
House pets (yes)		-0.001	-0.007	0.000	-0.01%
Rural-urban morphology					
	Urban	ref	ref	ref	ref
	Mixed rural-urban	0.089	-0.007	-0.001	0.82%
	Rural	0.114	-0.007	-0.001	1.06%
Residual				-0.007	
Overall EI				-0.076	

Table 6.6 shows the results for ever asthma at age 5. The contribution of income to overall income is 43%, the largest contributor. Other factors that contribute positively towards inequality are housing (16%), breastfeeding (11%), alcohol consumption (6%), smoking during pregnancy (5%), mother's mental health (5%) and mother's general health (2%) The residual is -0.005 and indicates unexplained variation (Table 6.6). Mode of delivery is a negative contributor (4.6%).

Table 6.6: Decomposition of the EI: Ever asthma age 5

	EI	**Elas**	**Cont**	**% Cont**
Ln income	0.033	-1.064	-0.035	42.51%
Child age	-0.003	0.152	0.000	0.55%
Child sex (male)	0.011	0.097	0.001	-1.24%
Birthweight				
Normal				
Low	-0.145	0.013	-0.002	2.33%
Very Low	-0.103	0.005	-0.001	0.69%
Family history of asthma	-0.006	0.119	-0.001	0.83%
Number of siblings				
No siblings				
1 sibling	0.044	-0.001	0.000	0.07%
2 siblings	-0.155	-0.010	0.002	-1.97%
3 or more siblings	-0.417	-0.005	0.002	-2.30%
Mother's education:				
No qualification				
NVQ level 1	-0.129	-0.021	0.003	-3.27%
NVQ level 2	0.006	-0.014	0.000	0.10%
NVQ level 3	0.359	-0.018	-0.007	7.93%
NVQ level 4	0.610	0.000	0.000	0.17%
NVQ level 5	-0.404	0.002	-0.001	0.74%
Overseas qualification	-0.602	0.001	0.000	0.61%
Housing				
Owned or mortgaged				
Council rented	-0.612	0.017	-0.010	12.35%
Rented privately	-0.419	0.008	-0.003	4.20%
Other housing	-0.718	-0.001	0.001	-0.66%
Mode of delivery				
Normal vaginal				
Assisted	0.129	0.015	0.002	-2.32%
Planned c-section	0.056	0.009	0.001	-0.61%
Emergency c-section	0.120	0.012	0.001	-1.71%
Other	-0.225	0.000	0.000	0.03%
Breastfeeding duration				
Never				
<2 months	-0.013	-0.008	0.000	-0.12%
2-4 months	0.148	-0.010	-0.001	1.75%
4-6 months	0.279	-0.007	-0.002	2.41%

	EI	Elas	Cont	% Cont
>6 months	0.156	-0.035	-0.005	6.61%
Mother's general health				
Excellent or good				
Fair or poor	-0.271	0.006	-0.002	2.12%
Mother's mental health				
Low distress				
Medium distress	-0.131	0.013	-0.002	2.02%
High distress	-0.495	0.004	-0.002	2.46%
Alcohol consumption				
None				
Light	0.191	-0.023	-0.004	5.31%
Heavy	0.062	-0.007	0.000	0.55%
Smoking during pregnancy				
Never smoked				
Gave up smoking	-0.055	0.005	0.000	0.30%
Kept smoking	-0.378	0.011	-0.004	5.05%
Mother's BMI				
Normal				
Low	0.070	-0.009	-0.001	0.77%
High	-0.017	0.002	0.000	0.05%
Smoke around child (yes)	-0.426	0.001	-0.001	0.77%
Damp (yes)	-0.263	0.006	-0.002	1.95%
Fume exposure (yes)	-0.046	0.024	-0.001	1.34%
House pets (yes)	-0.002	-0.002	0.000	0.00%
Rural-urban morphology				
Urban				
Mixed rural-urban	0.092	-0.006	-0.001	0.65%
Rural	0.110	-0.004	0.000	0.55%
Residual			-0.005	
Overall EI			-0.082	

Table 6.7 presents results for ever asthma at age 7. Income (68%) and housing (27%) are the largest contributors. Other positive contributors are breastfeeding (10%), mother's general health (9%), alcohol consumption and smoking during pregnancy which contribute about 5% each, and mother's mental health (2%). Negative contributors are mother's education (-20%) and mode of delivery (-3%).

Table 6.7: Decomposition of the EI: Ever asthma age 7

	EI	Elas	Cont	% Cont
Ln income	0.032	-1.656	-0.053	68.31%
Child age	0.000	0.415	0.0001	0.02%
Child sex (male)	0.009	0.103	0.001	-1.20%
Birthweight				
Normal				
Low	-0.158	0.008	-0.001	1.68%
Very Low	-0.122	0.007	-0.001	1.08%

	EI	Elas	Cont	% Cont
Family history of asthma	-0.003	0.139	0.0001	0.53%
Number of siblings				
No siblings				
1 sibling	0.049	-0.013	-0.001	0.79%
2 siblings	-0.138	-0.016	0.002	-2.82%
3 or more siblings	-0.424	-0.003	0.001	-1.59%
Mother's education				
No qualification				
NVQ level 1	-0.106	-0.011	0.001	-1.55%
NVQ level 2	0.042	-0.014	-0.001	0.76%
NVQ level 3	0.392	0.012	0.005	-5.95%
NVQ level 4	0.635	0.003	0.002	-2.39%
NVQ level 5	-0.380	0.002	-0.001	0.93%
Overseas qualification	-0.591	-0.016	0.009	-11.90%
Housing				
Owned or mortgaged				
Council rented	-0.576	0.026	-0.015	19.60%
Rented privately	-0.393	0.014	-0.006	7.22%
Other housing	-0.666	0.000	0.0001	0.00%
Mode of delivery				
Normal vaginal				
Assisted	0.133	0.004	0.0001	-0.65%
Planned c-section	0.062	0.004	0.0001	-0.32%
Emergency c-section	0.116	0.010	0.0001	-1.53%
Other	-0.232	0.000	0.0001	-0.06%
Breastfeeding duration				
Never				
<2 months	0.002	-0.005	0.0001	0.01%
2-4 months	0.149	-0.010	-0.002	2.01%
4-6 months	0.291	-0.001	0.0001	0.47%
>6 months	0.180	-0.031	-0.006	7.26%
Mother's general health				
Excellent or good				
Fair or poor	-0.294	0.023	-0.007	8.79%
Mother's mental health				
Low distress				
Medium distress	-0.140	0.010	-0.001	1.72%
High distress	-0.521	0.001	-0.001	0.74%
Alcohol consumption				
None				
Light	0.198	-0.020	-0.004	5.26%
Heavy	0.056	-0.008	0.0001	0.57%
Smoking during pregnancy				
Never smoked				
Gave up smoking	-0.040	0.003	0.0001	0.16%
Kept smoking	-0.362	0.012	-0.004	5.41%
Mother's BMI				
Normal				
Low	0.071	0.036	0.003	-3.29%
High	-0.006	0.015	0.000	0.12%
Smoke around child	-0.407	-0.009	0.004	-5.00%
Damp (yes)	-0.261	0.006	-0.001	1.86%
Fume exposure (yes))	-0.046	0.024	-0.001	1.45%
House pets (yes)	0.011	-0.022	0.0001	0.32%
Rural-urban morphology				
Urban				
Mixed rural-urban	0.107	-0.002	0.0001	0.31%

		EI	Elas	Cont	% Cont
	Rural	0.125	-0.004	0.0001	0.62%
Residual				0.0001	
Overall EI				-0.077	

6.7.2.2. Wheezing in the last 12 months

Table 6.8 presents results for wheezing in the last 12 months at age 3. Income is a large contributor to inequality (17%). Housing is also a large contributor, contributing about 30% to inequality. Other positive contributors are breastfeeding (20%), mother's mental health (19%), smoking during pregnancy (10%) and mother's general health (6%). Negative contributors are mother's education (-11%) and mode of delivery (-8%).

Table 6.8: Decomposition of the Erreygers' Index: Wheezing in the last 12 months age 3

	EI	Elas	Cont	% Cont
Ln income	0.035	-0.273	-0.009	16.95%
Child age	0.001	0.389	0.0001	-0.77%
Child sex (male)	0.010	0.096	0.001	-1.70%
Birthweight				
Normal				
Low	-0.132	0.012	-0.002	2.72%
Very Low	-0.094	0.006	-0.001	0.94%
Family history of asthma	-0.002	0.110	0.0001	0.48%
Number of siblings				
No siblings				
1 sibling	0.032	0.038	0.001	-2.20%
2 siblings	-0.157	-0.007	0.001	-1.96%
3 or more siblings	-0.405	0.004	-0.002	2.99%
Mother's education				
No qualification				
NVQ level 1	-0.602	-0.006	0.004	-6.76%
NVQ level 2	-0.125	-0.007	0.001	-1.62%
NVQ level 3	0.004	-0.012	0.0001	0.09%
NVQ level 4	0.356	0.004	0.001	-2.37%
NVQ level 5	0.600	0.001	0.001	-1.35%
Overseas	-0.408	0.001	0.0001	0.65%
Housing				
Owned or mortgaged				
Council rented	-0.609	0.023	-0.014	24.86%
Rented privately	-0.420	0.007	-0.003	5.18%
Other housing	-0.750	-0.001	0.001	-1.35%
Mode of delivery				
Normal vaginal				
Assisted	0.145	0.010	0.001	-2.54%
Planned c-section	0.056	0.009	0.001	-0.92%

	EI	Elas	Cont	% Cont
Emergency c-section	0.124	0.018	0.002	-3.91%
Other delivery	-0.230	-0.001	0.0001	-0.23%
Breastfeeding duration				
Never				
<2 months	-0.012	-0.018	0.0001	-0.40%
2-4 months	0.150	-0.019	-0.003	4.99%
4-6 months	0.279	-0.013	-0.004	6.65%
>6 months	0.159	-0.029	-0.005	8.25%
Mother's general health				
Excellent or good				
Fair or poor	-0.265	0.013	-0.003	6.20%
Mother's mental health				
Low distress				
Medium distress	-0.123	0.034	-0.004	7.52%
High distress	-0.468	0.013	-0.006	10.60%
Alcohol consumption				
None				
Light	0.188	-0.003	-0.001	1.02%
Heavy	0.064	0.000	0.0001	-0.02%
Smoking during pregnancy				
Never smoked				
Gave up smoking	-0.051	0.003	0.0001	0.28%
Kept smoking	-0.375	0.015	-0.005	9.74%
Mother's BMI				
Normal				
Low	0.068	-0.031	-0.002	3.84%
High	-0.019	0.014	0.000	0.46%
Smoke around child (yes)	-0.429	0.005	-0.002	3.46%
Damp (yes)	-0.267	-0.010	0.003	-4.54%
Fume exposure (yes))	-0.046	0.026	-0.001	2.16%
House pets (yes)	-0.001	-0.011	0.0001	-0.03%
Rural-urban morphology				
Urban				
Mixed rural-urban	0.089	-0.011	-0.001	1.76%
Rural	0.114	-0.010	-0.001	1.95%
Residual			-0.005	
Overall EI			-0.056	

Table 6.9 presents results for wheezing in the last 12 months, at age 5. Income is the largest contributor to inequality, contributing 55%. Other positive contributors are mother's mental health (21%), housing (20%), breastfeeding (14%), alcohol consumption (7%), smoking during pregnancy (6%) and mother's general health (6%). Negative contributors were mother's education (-24%) and mode of delivery (-4%).

Table 6.9: Decomposing the Erreygers' Index: Wheezing in the last 12 months age 5

	EI	Elas	Cont	% Cont
Ln income	0.033	-0.962	-0.032	54.61%
Child age	-0.003	-0.276	0.001	-1.42%
Child sex (male)	0.011	0.095	0.001	-1.72%
Birthweight				
Normal				
Low	-0.145	0.013	-0.002	3.36%
Very Low	-0.103	0.004	0.000	0.66%
Family history of asthma	-0.006	0.087	-0.001	0.87%
Number of siblings				
No siblings				
1 sibling	0.044	0.003	0.000	-0.21%
2 siblings	-0.155	0.002	0.000	0.44%
3 or more siblings	-0.417	0.002	-0.001	1.22%
Mother's education				
No qualification				
NVQ level 1	-0.602	0.007	-0.004	7.25%
NVQ level 2	-0.129	0.034	-0.004	7.66%
NVQ level 3	0.006	0.007	0.000	-0.06%
NVQ level 4	0.359	0.051	0.018	-31.86%
NVQ level 5	0.610	0.009	0.005	-9.10%
Overseas qualification	-0.404	0.003	-0.001	2.27%
Housing				
Owned or mortgaged				
Council rented	-0.612	0.012	-0.008	13.23%
Rented privately	-0.419	0.010	-0.004	7.14%
Other housing	-0.718	-0.001	0.001	-0.95%
Mode of delivery				
Normal vaginal				
Assisted	0.129	0.002	0.000	-0.44%
Planned c-section	0.056	0.015	0.001	-1.47%
Emergency c-section	0.120	0.011	0.001	-2.27%
Other delivery	-0.225	-0.001	0.000	-0.20%
Breastfeeding duration				
Never				
<2 months	-0.013	-0.006	0.000	-0.13%
2-4 months	0.148	-0.009	-0.001	2.39%
4-6 months	0.279	-0.005	-0.001	2.58%
>6 months	0.156	-0.033	-0.005	8.87%
Mother's general health				
Excellent or good				
Fair or poor	-0.271	0.012	-0.003	5.70%
Mother's mental health:				
Mother's mental health				
Low distress				
Medium distress	-0.131	0.041	-0.005	9.31%
High distress	-0.495	0.014	-0.007	12.29%
Alcohol consumption				
None				
Light	0.191	-0.018	-0.003	5.91%
Heavy	0.062	-0.009	-0.001	0.92%
Smoking during pregnancy				
Never smoked				
Gave up smoking	-0.055	0.004	0.000	0.40%
Kept smoking	-0.378	0.009	-0.003	6.00%
Mother's BMI				

	EI	Elas	Cont	% Cont
Normal				
Low	0.070	0.053	0.004	-6.47%
High	-0.017	0.022	0.000	0.65%
Smoke around child (yes)	-0.426	-0.006	0.002	-4.06%
Damp (yes)	-0.263	0.001	0.000	0.38%
Fume exposure (yes))	-0.046	0.015	-0.001	1.19%
House pets (yes)	-0.002	-0.004	0.000	-0.01%
Rural-urban morphology				
Urban				
Mixed rural-urban	0.092	-0.008	-0.001	1.29%
Rural	0.110	-0.006	-0.001	1.22%
Residual			-0.002	
Overall EI			-0.058	

Table 6.10 presents results at age 7. Income contributes the largest proportion towards inequality, 64%. Other factors that contribute positively are, poor general health in mothers (20%), mother's mental health (13%), housing (8%), alcohol consumption during pregnancy (8%), and smoking during pregnancy (6%). Negative contributors are mother's education (-24%), mode of delivery (-11%) and rural-urban morphology (-3%).

Table 6.10: Decomposition of the the Erreygers' Index: Wheezing in the last 12 months age 7

	EI	Elas	Cont	% Cont
Ln income	0.032	-0.610	-0.019	64.15%
Child age	0.000	0.026	0.000	0.00%
Child sex (male)	0.009	0.093	0.001	-2.77%
Birthweight				
Normal				
Low	-0.158	0.009	-0.001	4.61%
Very Low	-0.122	0.002	0.000	0.88%
Family history of asthma	-0.003	0.079	0.000	0.76%
Number of siblings				
No siblings				
1 sibling	0.049	0.001	0.000	-0.20%
2 siblings	-0.138	0.002	0.000	1.09%
3 or more siblings	-0.424	0.005	-0.002	6.70%
Mother's education				
No qualification				
NVQ level 1	-0.106	-0.010	0.001	-3.35%
NVQ level 2	0.042	-0.012	0.000	1.60%
NVQ level 3	0.392	0.004	0.002	-5.63%
NVQ level 4	0.635	0.003	0.002	-6.43%
NVQ level 5	-0.380	0.001	0.000	0.97%
Overseas qualification	-0.591	-0.006	0.003	-10.90%
Housing				
Owned or mortgaged				
Council rented	-0.576	0.014	-0.008	26.60%

	EI	Elas	Cont	% Cont
Rented privately	-0.393	-0.020	0.008	-25.85%
Other housing	-0.666	0.003	-0.002	7.57%
Mode of delivery				
Normal vaginal				
Assisted	0.133	0.011	0.001	-4.86%
Planned c-section	0.062	0.004	0.000	-0.73%
Emergency c-section	0.116	0.014	0.002	-5.34%
Other delivery	-0.232	0.000	0.000	0.09%
Breastfeeding duration				
Never				
<2 months	0.002	-0.006	0.000	0.04%
2-4 months	0.149	-0.011	-0.002	5.39%
4-6 months	0.291	-0.001	0.000	1.31%
>6 months	0.180	-0.006	-0.001	3.82%
Mother's general health				
Excellent or good				
Fair or poor	-0.294	0.021	-0.006	20.18%
Mother's mental health				
Low distress				
Medium distress	-0.140	0.022	-0.003	10.01%
High distress	-0.521	0.001	-0.001	2.57%
Alcohol consumption				
None				
Light	0.198	-0.010	-0.002	6.29%
Heavy	0.056	-0.008	0.000	1.48%
Smoking during pregnancy				
Never smoked				
Gave up smoking	-0.040	0.007	0.000	0.95%
Kept smoking	-0.362	0.004	-0.001	4.54%
Mother's BMI				
Normal				
Low	0.071	-0.022	-0.002	5.08%
High	-0.006	0.013	0.000	0.27%
Smoke around child (yes)	-0.407	-0.005	0.002	-6.18%
Damp (yes)	-0.261	0.000	0.000	-0.40%
Fume exposure (yes))	-0.046	0.001	0.000	0.22%
House pets (yes)	0.011	-0.022	0.000	0.79%
Rural-urban morphology				
Urban				
Mixed rural-urban	0.107	0.004	0.000	-1.54%
Rural	0.125	0.003	0.000	-1.41%
Residual			0.001	
Overall EI			-0.03	

At age 11 results are presented in Table 6.11. Income contributes towards 48% of overall inequality. Other large contributors are mother's mental health (40%), mother's general health (34%), breastfeeding (28%), housing (23%), mother's BMI (20%), smoking in the same room as the child (18%) and smoking during pregnancy (12%). Negative contributors are mother's education (20%) and mode of delivery (-12%).

Table 6.11: Decomposition of the Erreygers' Index: Wheezing in the last 12 months age 11

	EI	Elas	Cont	% Cont
Ln income	0.029	-0.266	-0.008	47.85%
Child age	0.000	0.711	0.000	2.10%
Child sex (male)	0.006	0.082	0.001	-3.16%
Birthweight				
Normal				
Low	-0.162	0.002	0.000	2.40%
Very Low	-0.136	0.001	0.000	0.66%
Family history of asthma	-0.007	0.078	-0.001	3.17%
Number of siblings				
No siblings				
1 sibling	0.054	-0.016	-0.001	5.48%
2 siblings	-0.137	-0.007	0.001	-5.79%
3 or more siblings	-0.429	0.002	-0.001	6.40%
Mother's education				
No qualification				
NVQ level 1	-0.601	0.012	-0.007	13.04%
NVQ level 2	-0.098	0.020	-0.002	11.96%
NVQ level 3	0.065	-0.002	0.000	0.70%
NVQ level 4	0.438	0.041	0.003	-20.67%
NVQ level 5	0.692	0.006	0.004	-27.45%
Overseas qualification	-0.384	0.001	0.000	2.75%
Housing				
Owned or mortgaged				
Council rented	-0.577	-0.003	0.002	-10.12%
Rented privately	-0.393	0.008	-0.003	19.19%
Other housing	-0.586	0.004	-0.002	14.20%
Mode of delivery				
Normal vaginal				
Assisted	0.143	0.004	0.001	-3.79%
Planned c-section	0.074	0.007	0.001	-3.25%
Emergency c-section	0.128	0.005	0.001	-4.34%
Other delivery	-0.206	0.000	0.000	-0.08%
Breastfeeding duration				
Never				
<2 months	0.012	-0.002	0.000	0.11%
2-4 months	0.167	-0.005	-0.001	5.36%
4-6 months	0.306	0.010	0.003	-19.54%
>6 months	0.207	0.011	0.002	-13.54%
Mother's general health				
Excellent or good				
Fair or poor	-0.318	0.017	-0.006	34.14%
Mother's mental health				
Low distress				
Medium distress	-0.130	0.013	-0.002	10.21%
High distress	-0.488	0.010	-0.005	29.57%
Alcohol consumption				
None				
Light	0.207	-0.004	-0.001	5.52%
Heavy	0.040	0.000	0.000	0.08%
Smoking during pregnancy				
Never smoked				
Gave up smoking	-0.034	0.001	0.000	0.27%
Kept smoking	-0.364	-0.005	0.002	11.33%
Mother's BMI				
Normal				

	EI	Elas	Cont	% Cont
Low	0.071	-0.045	-0.003	19.82%
High	0.002	-0.002	0.000	0.02%
Smoke around child (yes)	-0.418	0.007	-0.003	17.74%
Damp (yes)	-0.260	0.006	-0.002	9.36%
Fume exposure (yes)	-0.042	0.023	-0.001	6.01%
House pets (yes)	0.018	-0.014	0.000	1.54%
Rural-urban morphology				
Urban				
Mixed rural-urban	0.126	0.000	0.000	0.13%
Rural	0.148	0.000	0.000	-0.35%
Residual			-0.002	
Overall EI			-0.016	

6.7.2.3. Asthma medication use

Table 6.12 presents results asthma medication use, at age 5. The majority of inequality in asthma medication is explained by income (64%). Housing is also a positive contributor to overall inequality (22%). Mother's maternal health contributes 19% and mother's general health contributes 5%. Negative contributors are mother's education (-37%) and mode of delivery (-8%).

Table 6.12: Decomposition of the Erreygers' Index: Asthma medication use age 5

	EI	Elas	Cont	% Cont
Ln income	0.033	-0.403	-0.013	63.81%
Child age (male)	-0.003	-0.279	0.001	-4.00%
Child sex	0.011	0.031	0.000	-1.56%
Birthweight				
Normal				
Low	-0.145	0.011	-0.002	7.39%
Very Low	-0.103	0.002	0.000	0.84%
Family history of asthma	-0.006	0.031	0.000	0.85%
Number of siblings				
No siblings				
1 sibling	0.044	-0.003	0.000	0.61%
2 siblings	-0.155	-0.005	0.001	-3.72%
3 or more siblings	-0.417	0.000	0.000	0.42%
Mother's education				
No qualification				
NVQ level 1	-0.602	0.000	0.000	-0.18%
NVQ level 2	-0.129	0.003	0.000	2.12%
NVQ level 3	0.006	0.000	0.000	0.00%
NVQ level 4	0.359	0.017	0.006	-29.96%

	EI	Elas	Cont	% Cont
NVQ level 5	0.610	0.005	0.003	-15.26%
Overseas qualification	-0.404	0.003	-0.001	5.46%
Housing				
Owned or mortgaged				
Council rented	-0.612	0.004	-0.002	10.79%
Rented privately	-0.419	0.005	-0.002	10.73%
Other housing	-0.718	0.000	0.000	-0.65%
Mode of delivery				
Normal vaginal				
Assisted	0.129	0.002	0.000	-1.52%
Planned c-section	0.056	0.006	0.000	-1.51%
Emergency c-section	0.120	0.008	0.001	-4.87%
Other delivery	-0.225	0.000	0.000	-0.16%
Breastfeeding duration				
Never				
<2 months	-0.013	0.003	0.000	0.16%
2-4 months	0.148	-0.001	0.000	0.48%
4-6 months	0.279	-0.003	-0.001	4.39%
>6 months	0.156	-0.009	-0.001	6.80%
Mother's general health				
Excellent or good				
Fair or poor	-0.271	0.004	-0.001	5.45%
Mother's mental health				
Low distress				
Medium distress	-0.131	0.015	-0.002	9.34%
High distress	-0.495	0.004	-0.002	9.16%
Alcohol consumption				
None				
Light alcohol	0.191	0.003	0.001	-3.06%
Heavy alcohol	0.062	-0.003	0.000	1.00%
Smoking during pregnancy				
Never smoked				
Gave up smoking	-0.055	-0.002	0.000	-0.57%
Kept smoking	-0.378	-0.001	0.000	-2.05%
Mother's BMI				
Normal				
Low	0.070	0.005	0.000	-1.78%
High	-0.017	-0.001	0.000	-0.08%
Smoke around child (yes)	-0.426	0.003	-0.001	5.24%
Damp (yes)	-0.263	0.003	-0.001	3.38%
Fume exposure (yes)	-0.046	0.015	-0.001	3.38%
House pets (yes)	-0.002	-0.002	0.000	-0.01%
Rural-urban morphology				
Urban				
Mixed rural-urban	0.092	-0.004	0.000	1.70%
Rural	0.110	-0.004	0.000	1.85%
Residual			-0.004	
Overall EI			-0.021	

Finally, Table 6.13 presents results for asthma medication use at age 7. Income contributes 76% to overall inequality. Housing is also a large contributor, contributing 30% on aggregate to

inequality. Other positive contributors are mother's general health (23%), alcohol consumption (15%), smoking during pregnancy (8%) and mother's mental health (7%). On aggregate, the major negative contributor to inequality was mother's education (-24%).

Table 6.13: Decomposition of the EI: Asthma medication use age 7

	EI	**Elas**	**Cont**	**% Cont**
Ln income	0.032	-0.306	-0.010	76.14%
Child age	0.000	0.211	0.000	0.05%
Child sex (male)	0.009	0.027	0.000	-1.91%
Birthweight				
Normal				
Low	-0.158	0.003	0.000	3.75%
Very Low	-0.122	0.003	0.000	2.54%
Family history of asthma	-0.003	0.046	0.000	1.05%
Number of siblings				
No siblings				
1 sibling	0.049	-0.003	0.000	1.07%
2 siblings	-0.138	-0.011	0.001	-11.32%
3 or more siblings	-0.424	0.002	-0.001	6.12%
Mother's education				
No qualification				
NVQ level 1	-0.591	0.013	-0.008	59.51%
NVQ level 2	-0.106	0.023	-0.002	19.18%
NVQ level 3	0.042	0.011	0.000	-3.42%
NVQ level 4	0.392	0.047	0.010	-76.92%
NVQ level 5	0.635	0.006	0.004	-31.82%
Overseas qualification	-0.380	0.003	-0.001	9.69%
Housing				
Owned of mortgaged				
Council rented	-0.576	0.007	-0.004	29.89%
Rented privately	-0.393	0.001	0.000	1.85%
Other housing	-0.666	0.000	0.000	-2.24%
Mode of delivery				
Normal vaginal				
Assisted	0.133	-0.003	0.000	3.26%
Planned c-section	0.062	0.000	0.000	0.00%
Emergency c-section	0.116	0.001	0.000	-0.66%
Other delivery	-0.232	0.000	0.000	-0.16%
Breastfeeding duration				
Never				
<2 months	0.002	-0.001	0.000	0.02%
2-4 months	0.149	-0.003	-0.001	4.02%
4-6 months	0.291	0.000	0.000	0.56%
>6 months	0.180	-0.006	-0.001	8.28%
Mother's general health				
Excellent or good				
Fair or poor	-0.294	0.010	-0.003	22.82%
Mother's mental health				
Low distress				
Medium distress	-0.140	0.009	-0.001	9.59%
High distress	-0.521	-0.001	0.000	-2.95%
Alcohol consumption				
None				

	EI	Elas	Cont	% Cont
Light	0.198	-0.007	-0.001	11.50%
Heavy	0.056	-0.007	0.000	2.88%
Smoking during pregnancy				
Never smoked				
Gave up smoking	-0.040	0.003	0.000	0.96%
Kept smoking	-0.362	0.002	-0.001	6.63%
Mother's BMI				
Normal				
Low	0.071	-0.001	0.000	0.74%
High	-0.006	0.001	0.000	0.05%
Smoke around child (yes)	-0.407	-0.003	0.001	-10.06%
Damp (yes)	-0.261	-0.004	0.001	-8.49%
Fume exposure (yes))	-0.046	0.003	0.000	0.97%
House pets (yes)	0.011	-0.016	0.000	1.33%
Rural-urban morphology				
Urban				
Mixed rural-urban	0.107	-0.001	0.000	0.76%
Rural	0.125	0.001	0.000	-0.69%
Residual			0.004	
Overall EI			-0.013	

6.8. Discussion

6.8.1. Summary of results

Across all outcomes and ages, overall EIs are negative indicating that there is income-related inequality in asthma-related outcomes in UK children. Furthermore, the inequality in asthma outcomes is to the detriment of children in low-income families, where the burden of disease is disproportionately concentrated. My results show that income-related inequality is strongest for ever asthma compared to wheezing in the last 12 months (more negative EIs). Income-related inequality in asthma medication use is even less. Analysing changes in the EI over time, in the manner of Wagstaff et al (2003), using repeated cross-sections showed no particular trend of strengthening or weakening of income-related inequality for all outcomes. Estimations of income-related inequality using the CI and WI were also negative, but larger in magnitude. This evidence highlights the need for policy interventions in the UK to tackle the causes of income-related inequality in health outcomes.

To complement the above results, the decomposition analyses pinpoints factors that are driving income-related inequality in asthma outcomes. For all ages and outcomes (in which there was a statistically significant EI), decomposition analysis showed that income is the largest contributor to overall inequality, at all ages analysed and across all health outcomes (ranging from 17% to 76%). The contribution of income is driven mainly by the elasticity of the health outcome with respect to income, indicating the sensitivity of the health outcome to income. The findings reveal that income itself is the most important factor leading to income-related inequality in asthma outcomes. Another major contributor is income-related inequality in housing, which explains from 8% to 37% of income-related inequality. Several mother and child health-related factors also contribute towards income-related inequality. These are breastfeeding duration, alcohol consumption and smoking during pregnancy and mother's general and mental health. In the case of breastfeeding duration, breastfeeding for two or more months was more distributed in high-income families. Income-related inequality in asthma outcomes could therefore be improved by increasing breastfeeding duration for mothers in low-income families. In terms of alcohol consumption and smoking during pregnancy, these behaviours contribute towards income-related inequality in asthma-related outcomes. Therefore reducing the prevalence of these behaviours through advocacy and health literacy could lead to not only a reduction in income-related inequality, but also improved asthma-related outcomes for all children, and particularly those in low-income families where the burden of disease falls. At all ages and outcomes, mother's education had an overall negative contribution to income-related inequality in asthma-related outcomes driven by large positive EI's for high NVQ levels (income-related inequality in mother's education in favour of high income families). This indicates that mothers with high levels of education are distributed disproportionately in high income families. Therefore, improving the education levels of mothers in low income families is likely to improve income-related inequality in asthma outcomes.

The range of key factors that contribute to income-related inequality in asthma outcomes, suggest that policies targeted at reducing income-related inequalities in health should involve a number of key players. Tackling chronic health problems, such as asthma and related outcomes, is a multi-level societal problem that would benefit from including not only governments, but also society and the communities burdened with adverse health outcomes. Potential policies could include improved housing allowances for low-income families and ensuring an adequate level of support can be obtained through the Universal Credit scheme. Policy recommendations are discussed in more detail in Chapter 7.

To the best of my knowledge there is no published literature on income-related inequality in asthma outcomes as measured by the EI (CI or WI) , nor decomposition analysis in this context. Therefore I cannot directly compare my findings to other studies, but have opened the way to a new research approach in the area of childhood asthma-related outcomes and hope that further research along these lines will follow.

6.8.2. Strengths and limitations

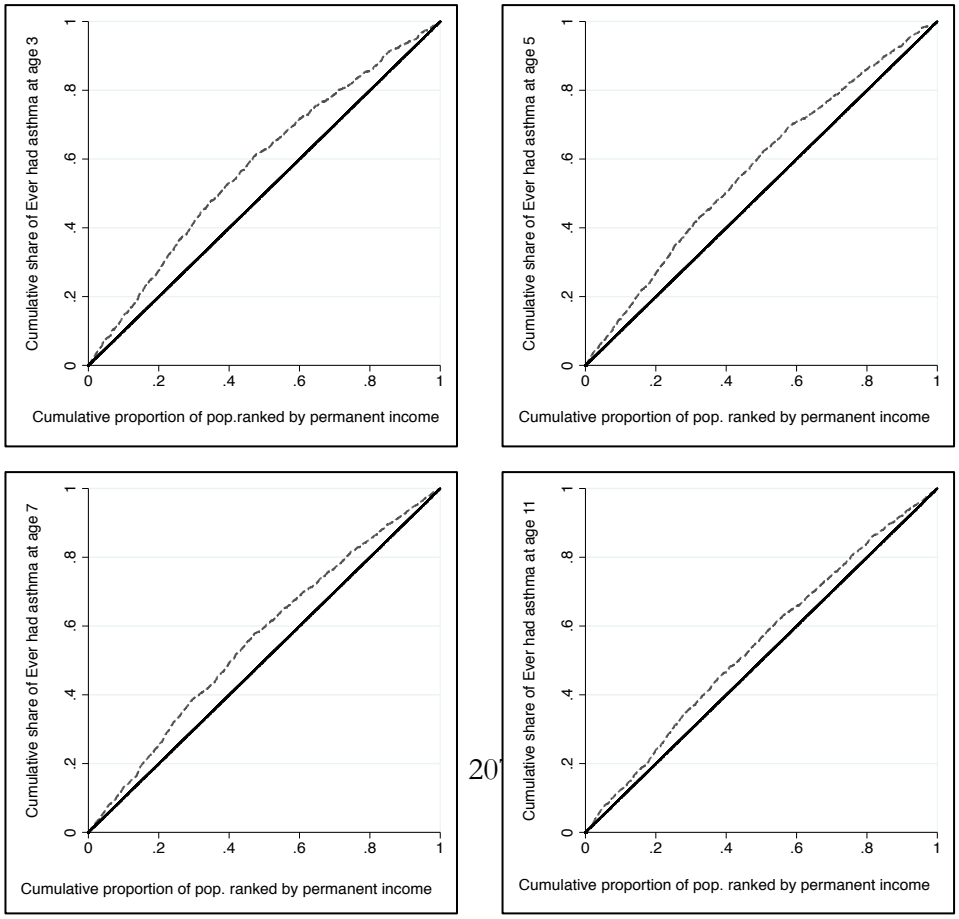
One limitation of the analysis is that the health outcomes are derived from subjective mother-reported responses to ever asthma, wheezing in the last 12 months and asthma medication use of their children which could result in biased results. Another limitation is that the study does not extend to explore the dynamics of the EI to explore whether changes in asthma-related outcomes have favoured children who initially had lower or higher income, i.e. assessing income mobility, which provides scope for future research.

Despite some limitations, the results of this chapter present the first ever attempt to study the extent of income-related inequalities across a range of asthma-related outcomes in UK children,

across several ages. The use of permanent income ensures that income-related inequality estimates reflect persistent low income (long-term) rather than transitory episodes of low income (short-term), which is a strength of the analysis. The results have the advantage of pinpointing factors which contribute to income-related inequality, that might not be picked up by standard regression techniques thus offering valuable insight for policy setting.

6.9. Conclusion

The results of this chapter showed that asthma-related outcomes in children are disproportionately concentrated amongst children from low-income families, across all ages. Decomposing inequality into contributing factors revealed that income is a major contributing factor to income-related inequality, but that other factors such as housing tenure and mother's health also contribute towards inequality, whereas mother's education reduced overall income-related inequality.



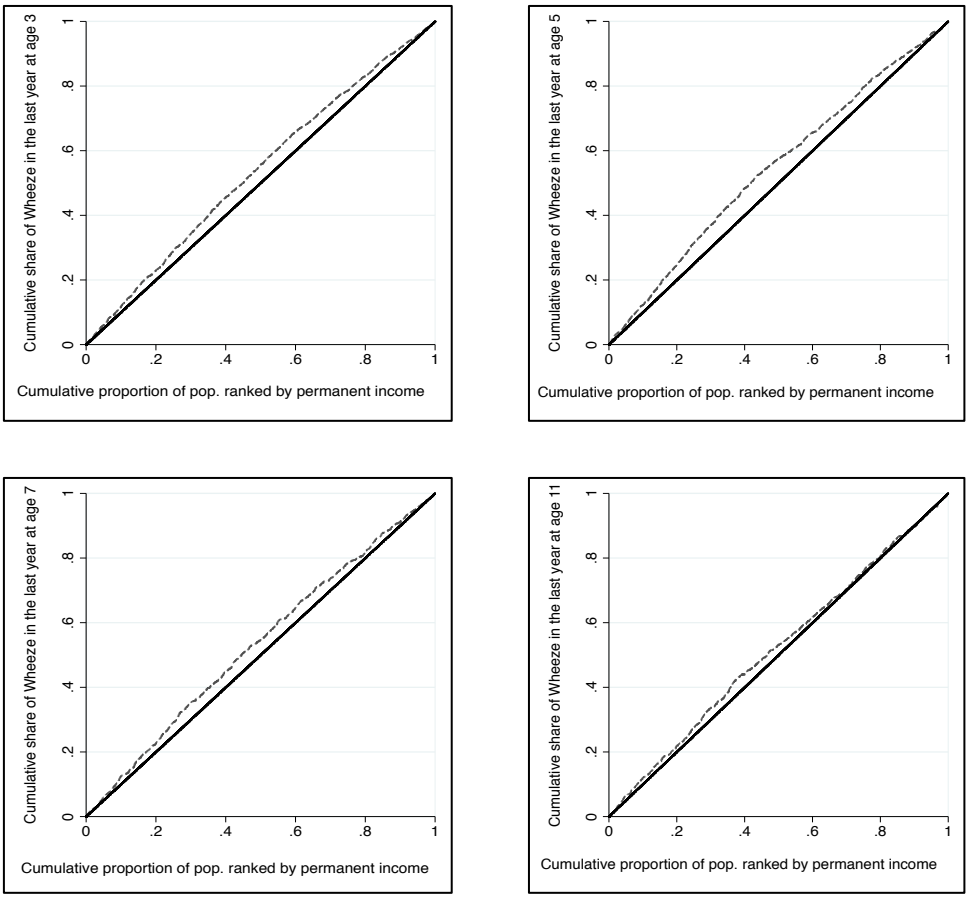


Figure 6.4: Concentration curves: Wheezing last 12 months

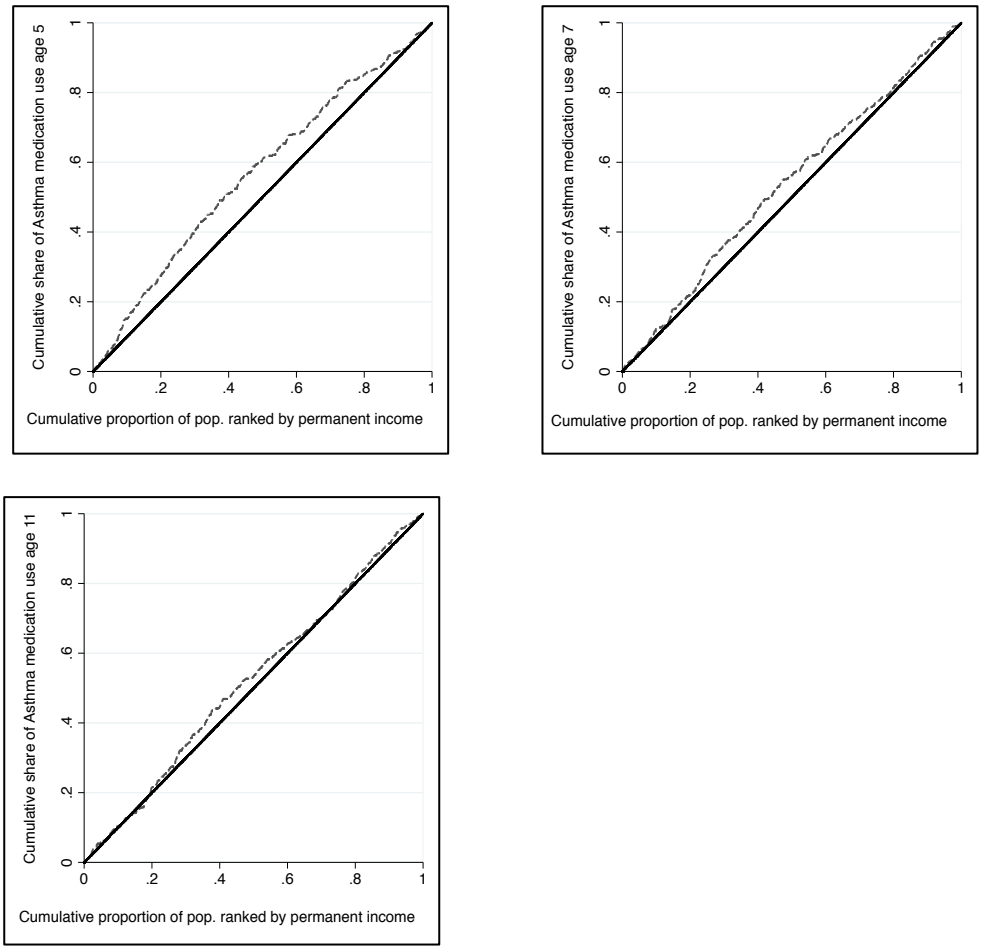


Figure 6.5: Concentration curves: Asthma medication use

7.Discussion of thesis

7.1. Introduction

The objectives of the thesis were to investigate, using a rich panel data set, the MCS, the association between family income and asthma-related outcomes in UK children (namely the child health-income gradient), and to assess whether it strengthens as children grow up. This association was assessed cross-sectionally (Chapter 3) as well as longitudinally, by classifying wheezing in children by age at onset and duration, into wheezing phenotypes (Chapter 4), better reflecting heterogeneity in wheezing. Panel data methods (fixed and random effects) were applied to further exploit the longitudinal nature of the data and address limitations of cross-sectional analysis (Chapter 5). Finally, taking into account the full socioeconomic distribution of children in the MCS, income-related inequality in asthma-related outcomes was estimated and decomposed into contributing factors (Chapter 6).

7.2. Review of findings

7.2.1. The association between income and asthma-related outcomes

Chapter 3 investigated the association between family income and asthma-related outcomes cross-sectionally. Analysis was performed on dichotomous measures derived from questions asked in the MCS, these were: ever asthma (ages 3, 5, 7, 11), wheezing in the last 12 months (ages 3, 5, 7, 11) and asthma medication use (ages 5, 7, 11). Family income was measured as the

natural logarithm of permanent income, inflated to 2011 prices. In order to adjust for other explanatory variables, a five stage step-wise approach was performed. For the outcome ever asthma, fully adjusted models showed a strong association with low income. A 1% increase in family income was associated with a 1.7% decrease in the probability of ever asthma at age 3, rising to a 7.2% decrease at age 11. There was also a statistically significant association between low income and wheezing in the last 12 months, which was statistically significant at ages 5, 7 and 11 but not at 3 years, in the fully adjusted models. A 1% increase in family income was associated with a 2.5-3% decrease in the probability of wheezing in the last 12 months, at ages 5 to 11. Finally, there was also a statistically significant association between low income and asthma medication use at ages 5, 7 and 11 years. A 1% increase in family income was associated with a 1.8% decrease in the probability of asthma medication use at age 5, rising to a 2.4% decrease at age 11.

As a sensitivity analysis, family income was measured as transient income. For all asthma-related outcomes, results with transient income showed a similar trend in terms of the direction of the association, but were smaller in magnitude. This supports the permanent income hypothesis that long-run income more accurately determines health investments, and therefore child asthma-related outcomes are more closely related to permanent income rather than short-term income (Blau, 1999; Case et al., 2002).

7.2.2. Steepening of the child health-income gradient

A secondary objective of the analysis in Chapter 3 was to assess if the income gradient in asthma-related outcomes changes with age. For child health generally, the evidence about this is inconclusive in the UK for general health or asthma-related outcomes (Currie et al., 2007; Apouey et al., 2013; Kuenhle, 2014).

The MCS data used in my thesis covers a relatively long follow-up, up to age 11, whereas the longest follow-up period in previous studies of income and asthma outcomes was until age 6. The results showed that there were statistically significant differences in the income gradient between ages 3 and 11, for the case of ever asthma, and between ages 5 and 11 for wheezing in the last 12 months and asthma medication use. These are novel results for the UK and demonstrate that the socioeconomic gradient in asthma-related outcomes strengthens as children grow older.

7.2.3. Income and wheezing phenotypes

One limitation of the cross-sectional analysis was the use of dichotomous outcomes and their limited ability to capture a complex disease, such as asthma and related symptoms. Chapter 4 exploited the longitudinal nature of the MCS data in this regard. I identified mutually exclusive wheezing phenotypes based on the time of onset and duration of wheezing in children, accounting for some of the heterogeneity in asthma outcomes. The results of the adjusted models in the base case showed that low income was only associated with late onset wheezing (marginal effect: -0.028), although this statistical relationship was weak. However, this evidence is consistent with the results for wheezing in the cross-sectional analysis which showed that income was not associated with wheezing at age 3, but it was at older ages. This once more emphasises the importance of addressing low income in the early years, to prevent ongoing asthma-related health issues. In the sensitivity analyses, the cut-off age for early remitting wheeze was altered to age 7 (from age 5 in the base case). The results based on this definition showed a statistically significant relationship between low income and persistent wheezing (marginal effect: -0.02). Overall, the results of the phenotype analysis highlighted that low-income children are at higher risk of late onset wheezing and persistent wheezing, which may

both persist into adult life. These findings demonstrates the importance of distinguishing between phenotypes and their determinants, to be able to implement policy effectively.

7.2.4. Longitudinal analysis using panel data methods

The cross-sectional analysis treated each MCS survey as an independent data set; it did not link the repeated measures of income and asthma-related outcomes. On the other hand, Chapter 4 used the longitudinal wheezing outcomes to categorise wheezing into phenotypes, but did not exploit the variation in family income across time. Panel data methods applied in Chapter 5 allowed me to investigate how family income influences asthma-related outcomes for the same individuals over an eight year period. Panel data methods provide more accurate estimations due to more variability in the data, as they consider both between and within variation in income and child health outcomes. In this way, they are more likely to infer causality. Both Fixed Effects (FE) and Random Effects (RE) models were applied to the data, with the Hausman test being used to select the more suitable estimation.

FE model provided consistent and efficient estimators for outcomes ever asthma and wheezing in the last 12 months. However, for the outcome ever asthma, this association was weak, and for wheezing in the last 12 months the association was not statistically significant. The RE model provided a consistent and efficient estimator for asthma medication use, but family income was weakly associated with the outcome. Generally, the results show that income has a weakly causal effect on asthma-related outcomes. This implies that the results of the cross-sectional analysis, whilst providing strong evidence for the association between income and asthma-related outcomes at each age, need to be interpreted with caution. As discussed in Chapter 3, there are other underlying factors that lie on the causal pathway between income and asthma related outcomes, which drive the variation in health outcomes in children.

A second longitudinal approach, Approach 2, was used in Chapter 5. It considered interaction terms between income and MCS survey age. Pooled data was used to account for software limitations in Approach 1, by applying survey elements such as clustering. The results of Approach 2 were comparable to the cross-sectional results, with statistically significant associations between family income and asthma-related outcomes. However they were subject to the same caveats as the cross-sectional analysis in terms of inferring causality.

7.2.5. Income-related inequality in asthma-related outcomes

The overarching conclusion from Chapters 3, 4 and 5, confirms that low income is a key determinant of asthma-related outcomes. However, these types of analyses do not provide a measurement and explanation of inequality in child asthma-related outcomes across the entire distribution of family income. To assess this, I estimated Erreygers' Indices (EIs), which are an adapted form of the Concentration Index (CI). Using the MCS data, results showed negative EIs across all ages and outcomes, indicating that adverse asthma-related outcomes were more concentrated in children from low-income households. On analysis of the concentration curves, income-related inequality appeared to decrease with age (less negative EIs at each age and outcome), which could be due to children from low-income families recovering from health shocks over time, and having better outcomes in the long-run. Decomposition analysis revealed that the main contributor (19% to 76%) to overall income-inequality in all asthma-related outcomes was income itself, driven mainly by its elasticity.

The results of Chapter 6 complement findings from the previous chapters by quantifying the extent of income-related inequalities in child asthma-related outcomes, and showing that in the UK, ill-health in children is more concentrated amongst children in low-income families. This is in line with data that shows the unequal spread of income in the UK, and adds to the literature

– that not only is income unevenly distributed in the population, but that this uneven spread contributes to a higher burden of asthma in children from low-income families (OECD, 2019). The findings from estimations of income-related inequality in asthma outcomes and decomposition analysis are novel in the context of the UK.

7.2.6. Summary of all results

Comparing the results across all the methodological chapters of this thesis indicates that the findings across chapters are complementary – that whether measured cross-sectionally at each age, or longitudinally considering all ages, the relationship between income and a number of asthma-related outcomes persists, to some degree. It shows that using more nuanced measures of wheezing, such as wheezing phenotypes, may improve the predictability of specific asthma-related outcomes that result from low income. Additionally, measures of income-related inequality in these outcomes indicate that there is an unequal distribution of asthma-related outcomes in UK children, with poor children being at a disadvantage. Combined with evidence that already exists on the adverse effect of income on child health, UK policy makers must consider the abundance of evidence in support of welfare for children in low-income families, as well as renewed funding towards child health programmes within public health agendas.

7.3. Comparison with other studies

The magnitude and direction of the association between income and ever asthma and wheezing as observed in my analyses are comparable to previous findings in the UK for ages 3 and 5 years, as in Violato et al (2009). However, my analysis was the first to include children up to age 11, in the context of the income and asthma-related outcomes in the UK, showing some evidence of steepening of the child health-income gradient, which was not evident in the Currie

et al (2007), Apouey et al (2013) or Kuehnle (2014) studies, in the context of general health, and Violato et al 2009, in the context of asthma-related outcomes.

In terms of the sensitivity analysis where transient income was used in place of permanent income, the results showed that estimated coefficients for ever asthma and wheezing in the last 12 months were larger when permanent income was used in the models. This follows other studies that have implemented both measures of income and supports the hypothesis that long-term income has more power to determine outcomes than transient income (Case et al., 2002; Propper et al., 2006; Violato et al., 2009).

In the analysis of income and wheezing phenotypes (Chapter 4), the results compare well to studies that have also investigated the link between socioeconomic status and wheezing phenotype, which also found statistically significant associations between low socioeconomic status and persistent wheezing in children (Panico et al., 2014; Galobardes et al., 2015; Taylor-Robinson et al., 2015). Comparison for the late onset wheezing phenotype is however limited, because the Taylor-Robinson et al (2015) and Panico et al (2014) studies did not include a late onset wheezing phenotype; the Galobardes et al (2015) study used paternal education level as their measures of socioeconomic status. In this respect my results are novel in that they indicate that low income increases the chances of a child having wheezing that begins only after age five and not before. However, this effect was relatively small and only weakly statistically significant.

Previous studies have used measures of inequality to assess the income-related inequality of a specific health measure within a population, but in high income countries this research is limited to general health or obesity in children (Van Doorslaer and Koolman 2004; Walsh and Cullinan, 2015). My thesis presented negative and statistically significant Erreygers' Indices, which indicate the unequal distribution of asthma-related outcomes in UK children for the first time.

To my knowledge, decomposition analysis has not been performed in the context of asthma-related outcomes.

7.4. The influence of other variables

Aside from the direct relationship between income and asthma-related outcomes described, the results of this thesis showed that the variation in asthma-related outcomes is also explained by the other explanatory factors that were included in the analyses. These factors may act as transmission mechanisms and attenuate the direct association between income and asthma-related outcomes. Most of the explained variation was from mother and child health-related factors, which included breastfeeding duration, mode of delivery, mother's general health and mother's mental health.

Specifically, statistically significant associations between breastfeeding duration and asthma-related outcomes indicated that children who were breastfed for two or more months had a lower risk of asthma-related outcomes compared to children who were never breastfed or breastfed for less than two months. The protective effect of breastfeeding appeared to last throughout childhood from ages 3 years to 11 years and supports the extensive literature that provides evidence on the positive health benefits associated with breastfeeding (Pokhrel et al., 2015).

In terms of mode of delivery, children born by caesarean section were also more likely to have asthma-related outcomes at all ages, compared to children born by normal vaginal delivery. This result may be explained by the hygiene hypothesis regarding lack of contact between the infant and mother's bacteria, which are necessary for immune development (Azad et al., 2013). Alternatively, it could be that the maternal or foetal condition which necessitated a caesarean

birth may have also affected immune function or lung development in the baby, which in turn increased the risk of asthma-related outcomes (Darabi et al., 2019). My results highlight that caesarean birth may be associated with adverse outcomes in the child and that this intervention should only be used when absolutely necessary, rather than optional (Darabi et al., 2019).

Mother's health (both general and mental health) was found to be an important determinant of asthma-related outcomes in children. An explanation could be that the quality of care provided to children by mothers who are generally ill or who suffer with mental health illness over a prolonged period of time, tends to be of a poorer quality than mothers who are healthy, as explained by the 'good parent' theory of Mayer (2002). This emphasises the importance of social care and support systems for parents, especially mothers who are vulnerable to mental health problems during and after pregnancy, and whose well-being may have a spill-over effect on their children.

In terms of the inequality and decomposition analysis, determinant factors that contributed substantially to income-related inequality in asthma outcomes, other than income, included housing tenure which was the next largest contributor to inequality (up to 37%), after income. This indicates that because housing tenure is a significant driver of asthma-related inequalities in childhood, provision of adequate housing for low-income families is important to reduce income-related inequality in asthma outcomes. Mother's general and mental health and breastfeeding duration also contributed towards inequality across all asthma-related outcomes. Mother's education had the opposite effect and reduced overall income-related inequality. The results of the decomposition analysis offer policy makers an indication of what areas and factors can be targeted in order to improve socioeconomic inequality in asthma-related outcomes in children.

7.5. Strengths and limitations of the thesis

This thesis provides strong evidence to support the importance of family income as a determinant of asthma-related outcomes in UK children. It uses a longer follow-up than has previously been used in similar studies (Violato et al., 2009), offering new insight. It also exploits the panel nature of the MCS data, adding to the body of economic literature on family income and child health that generally uses cross-sectional analysis.

In terms of the inequality analysis, the quantification of income-related inequality in asthma-related outcomes and the subsequent decomposition analysis, is a novel investigation in the case of UK children. Studies of income-related inequality in health outcomes are becoming increasingly important in the context of the UK as income-related inequality is increasing (OECD 2019). This thesis transforms standard measures of income-related inequality and subsequent decomposition analysis into tools for policy evaluation in the UK healthcare system.

In terms of limitations, the main outcomes in the analyses of this thesis have been derived from MCS questions which were asked of the main respondent. Although these questions are validated questions that have been used broadly in the epidemiological and economic literature on asthma, the issue of subjectivity and recall bias remains. Objective data, for example, direct diagnoses from medical professional or physiological tests such as peak respiratory flow, would be ideal to overcome some of these limitations. However, the MCS does not include objective health data of this nature, as it would make such cohort studies very costly and difficult to follow-up. I have used three different measures of asthma-related outcomes in this thesis to help ensure results are robust.

Another limitation is that a causal link between income and asthma-related outcomes cannot be inferred by cross-sectional analysis. The extensiveness of the MCS fortunately allowed me

to control for numerous variables, which removed biases in the estimation of the impact of income. However, it is possible that there still remain unobserved factors driving the variation in asthma-related outcomes. One potential example is environmental pollution or air quality – specifically, children in low-income families may be more likely to live in polluted areas, and this, rather than low income itself, may explain the increased risk of asthma-related outcomes. However, given the extensive nature of variables included in the analysis, it is likely that they account for some of the unobserved variables, specifically rural-urban morphology is likely to be a proxy for environmental pollution.

Nonetheless this thesis was intended to be an explorative analysis, employing a series of complementary methodological techniques including quantifying income-related inequality in asthma-related outcomes. The statistically significant associations found, provide insightful evidence into the relationship of income and other factors, on asthma-related outcomes in children in the UK.

7.6. Policy implications

7.6.1. Income welfare

The results of this thesis suggest that income is strongly associated with asthma-related outcomes. Even when taking into account a large number of other explanatory factors and adjusting for those, the association between income and asthma-related outcomes remains. It is possible that there are other factors that I have not adjusted for, and that the association between income and these outcomes may be at least partly explained by residual confounding effects. However, this may also support what was indicated by Cooper and Stewart (2020), i.e.

that poorer children have worse outcomes *because* they are poor and this is not totally attributed to other determinant factors that are also associated with income.

The association may also be explained by considering that income alone cannot directly contribute to health, but rather acts as a channel through which good health can be achieved. Then this indicates that there are underlying mechanisms through which income influences health. For asthma-related outcomes, these mechanisms may include pregnancy and birth-related factors such a smoking in pregnancy, low birthweight, mode of delivery and breastfeeding, all of which I have attempted to adjust for in my analyses and that are all socioeconomically patterned. Other potential mechanisms may be related to childhood infections, nutrition and growth in early childhood, or the environment, such as pollution.

In the UK, the NHS[43] provides free access at the point of service and children's prescriptions such as asthma medications, are also free. Therefore, a final potential explanation is that low-income families have different health seeking behaviour compared to high income families. There is evidence to show that low-income families are less likely to seek health care in the early stages of illness or to seek preventative health care (Cookson et al., 2016). It is therefore possible that some of the excess variation of asthma-related outcomes could be due to poorly managed early symptoms of asthma. Such health seeking behaviour could be driven by the opportunity cost that comes with seeking out health care, for example missed hours at work. It could also be due to the time constraint low-income families may face. There are a few potential mechanisms behind my findings, which point to why low-income families need income support through adequate welfare policies, to improve the health outcomes of their children.

[43] This refers to NHS England, Scotland and Wales; and Health and Social Care in Northern Ireland

In 2010, almost coinciding with the final year analysed in this research (2011), child poverty was at a 13-year low, a prevalence of 27% of UK children (Brewer et al., 2011). This coincided with policies implemented by the Labour government from the mid-1990s. These policies included the introduction of a minimum wage, establishment of a more progressive tax and benefit system, and protection of and increases in benefits targeted specifically at children (CPAG, 2017). Policies also included measures aimed at encouraging parental employment, particularly single parents, such as the New Deal for Lone Parents program. Furthermore, the 2010 Child Poverty Act had just been passed as law, committing the new government to further poverty reduction over the coming decade. There is also evidence to support that state benefits helped to increase the disposable income of the poorest families in the UK, showing that between 1998 and 2010, half a million children were lifted out of poverty (Marmot, 2010).

However, due to reform and austerity measures implemented since 2010, the UK has seen fewer children being lifted out of poverty (and an overall increase to 30% of children living in poverty by 2016) (Francis-Devine, 2020). The Welfare Reform and Work Act 2016 resulted in a decrease in the amount of income support available to families, by lowering the household benefit cap from £26,000 to £20,000. It also introduced a four year benefits freeze between 2016-2020, which has seen benefits remain constant, despite increases to the inflation rate (Hood and Waters, 2017). Today, benefit cuts have left children from low-income families more vulnerable than ever before, and therefore at risk for adverse health outcomes, especially chronic illnesses such as asthma. The UK is in need of pro-poor welfare reform to ensure that the most vulnerable in society are protected. Reducing child poverty and the adverse impact that poverty has on child health is not only morally, but legally the right thing to do, across all societies including the UK (Marmot, 2020). The current government seeks to reduce child poverty through encouraging parents to work and earn a salary, rather than through tax credits and

benefits. Whilst promoting employment through training and job creation, the government may need to consider strategies to also raise the wages of low-paid workers, as this would make employment an economically attractive choice. This could be done either through better hourly pay or by improving their prospects of progression to a higher pay grade. The high costs that come with working, namely the cost of childcare and transport, are also issues that will have to be addressed to incentivise parents to work whilst ensuring that they can in fact afford to work. Essentially, families must be able to achieve a minimum income for healthy living, which includes the cost of purchasing items that are needed to ensure adequate levels of nutrition, physical activity, housing, psycho-social interaction, clothing, transport, heating and hygiene (Hirsch, 2019). To this effect, the financial welfare for low-income families should at least increase in line with inflation, and there should be a higher cap on benefits that is based on a minimum income for healthy living.

7.6.2. Mother's health

7.6.2.1. Mother's general health

In the UK where the cost of healthcare is not a barrier to health, policies such as improving health literacy in mothers, may be key to improving their health outcomes. Health literacy, which includes access to reliable health information at the community level and from GP practices, has been shown to be one of the strongest determinants of ill-health – stronger than education level, deprivation, age or ethnicity (WHO, 2020). NHS England[44] has set up a national collaboration that includes Public Health England (PHE) and Health Education England, to take a systems approach to tackling health literacy. Effective programmes have been

[44] Public Health Wales Strategic Plan 2019-2022 includes greater health literacy; NHS Scotland has also published a health literacy plan: 'Making it Easier. A Health Action Literacy Plan for Scotland. 2017-2025'

implemented in the East Midlands, with a focus on diabetes in the deprived groups (PHE, 2015). The programme has so far proved successful and the UK would benefit from a country-wide introduction, and for a wider range of conditions. Improving mother's health through health literacy, would then place them in a better position to provide care and support for their children, especially when a child requires ongoing care for chronic conditions such as asthma.

Additionally, policy for mother's health should be aimed at a more holistic health approach for women, with less fragmentation in the health system. The Royal College of Obstetrics and Gynaecology (RCOG) is calling for the creation of a national strategy for women's health based on a life course approach (RCOG, 2019). This will help the UK to move away from a disease intervention service provision, towards a more preventive health service, ensuring that women's health and wellbeing becomes a priority throughout their lives (RCOG, 2019). There are currently no specific policies aimed at advocating for health of all mothers in the UK, with the exception of new mothers and their babies. Investment in mother's health throughout their lives, is likely to have long-run benefits and positive spill-over effects onto their children, improving health outcomes.

7.6.2.2. Mother's mental health

In 2012, the former coalition government in the UK put in place the Health and Social Care Act (2012)[45] that, for the first time, recognised the government's duty towards both physical and mental health of the population. Furthermore, the 2015 Government's Mandate to NHS England stated that NHS England's objective is to put mental health on a par with physical health, a concept known as parity of esteem. The current Conservative government has since

[45] Some provisions of the Health and Social Care Act 2012 extend to England, Northern Ireland, Scotland and Wales.

set objectives for parity of esteem in successive mandates with the NHS Five Year Forward View, calling for parity by 2020 (Parkin, 2021). Scotland has also committed to parity of esteem in its Mental Health Strategy 2017-2022, and follows its own legislature on mental health, namely the Mental Health Act (2003) [46]. Although mental health has only in the last decade begun to receive the recognition it requires, this is a step in the right direction towards providing mothers with all the health services they might need. My results showed that mother's mental health is associated with adverse asthma-related outcomes in children, therefore health care provision for mother's with mental health conditions is likely to improve child health asthma-related outcomes.

7.6.3. Breastfeeding in the UK

My results showed that breastfeeding for any duration of time, compared to never breastfeeding is associated with better asthma-related outcomes. The health benefits of breastfeeding on child health have been well documented – including the protective effect against illnesses such as asthma (Azad et al., 2017). However, the UK still has one of the lowest rates of breastfeeding in Europe, with mothers from deprived backgrounds being less likely to breastfeed their children (RCPCH, 2019). At six months, only 34% of babies in the UK are receiving some breast milk compared to 49% in the US, and 71% in Norway. Evidence has shown that supporting mothers who are exclusively breast feeding one week after birth, to continue breastfeeding until their baby is four months, could save the NHS at least £11 million annually (Pokhrel et al., 2015). Additionally, doubling the proportion of mothers breast feeding for seven

[46] The Welsh government has published the 'Together for Mental Health: A Strategy for Mental Health and Wellbeing in Wales' which is a 10-year strategy for improving the lives of people using mental health services, their carers and their families; The Department of Health in Northern Ireland has drafted their first ever 10 year Mental Health Strategy.

to 18 months could save £31 million, by reducing breast cancer and increasing both quantity and quality of life for mother and child (Pokhrel et al., 2015).

Policy to improve breastfeeding uptake should have a special focus on mothers from deprived backgrounds, as children in low-income families are already faced with other factors that put them at risk of poor health outcomes. Policy should also aim to address the financial, practical and social constraints that low-income mothers face when it comes to breastfeeding, for example, taking time off during the working day to breastfeed. Potential policy recommendations include introducing a national strategy to promote the initiation and continuation of breastfeeding in the UK (RCPCH, 2019). Such a strategy would ideally include different stakeholders such as the government and employers, as breastfeeding support is very much a societal issue, rather than the sole responsibility of mothers. The national strategy could also include the re-introduction of a nationwide survey to monitor infant feeding and provide an evidence base for breastfeeding. Data is key for driving policy and for monitoring and evaluation purposes, therefore a nationwide survey has the potential to provide invaluable insight to governments and other health bodies (Rollins et al., 2016).[47] Currently in the UK, the RCPCH has called on national agencies in England, Scotland, Wales and Northern Ireland to develop breastfeeding strategies using a multi-disciplinary approach, supported by sound evidence. This is further evidence of the lack of an existing and coherent breastfeeding strategy in the UK, highlighting the urgency of addressing the issue.

[47] The UK Infant Feeding Survey used to be conducted every 5 years between 1975 and 2010 but the survey was cancelled in 2015 and has not been conducted since.

7.7. Further research

There are a number of research recommendations following on from my thesis. Conducting follow-up analyses using new MCS data[48] that has become available since undertaking this research, could provide more insight. The MCS children have now reached adolescence and are more likely to have a true diagnosis of asthma or remitted from childhood wheezing. Inclusion of these additional ages could strengthen the panel data analysis by potentially offering more variability in family income and asthma-related outcome data. Longer follow-up data would also allow one to assess whether the family income-child health gradient steepens into adolescence.

Although this thesis has explored the association between income and asthma-related outcomes quantitively, the explanations for the evidence provided from the results is somewhat speculative. Qualitative analyses could provide a more in-depth understanding into some of the behavioural aspects occurring in the home, that may translate into adverse asthma-related outcomes seen in low-income families. Qualitative analyses can complement quantitative analyses by enabling a more nuanced explanation of the experiences, causes and mechanisms of an effect, which is not possible to gather using only quantitative survey instruments.

In terms of adverse asthma-related outcomes in the UK, poor air quality has been identified as a major cause (Cakmak et al., 2016). In the UK it has been difficult to include data on air pollution in analysis as it not readily linked to data on child health. However, the CLS has recently linked the MCS with data on air pollution, from the Multiple Environmental

[48] i.e. the age 14 (MCS 6) and age 17 (MCS 7). Data on asthma-related outcomes in the new surveys is limited to only one of the three outcomes used in this thesis, and it phrased differently. The variable is derived from the question "Does the child have asthma?" rather than "Has the child ever had asthma".

Deprivation Index (MEDIx). This data contains mean concentrations of carbon monoxide, particulate matter, nitrogen dioxide and sulphur dioxide (Midouhas et al., 2019). Including pollution data in my analysis could provide useful insights into the impact of air pollution on asthma and wheezing outcomes in children.

In terms of analysing the income-related inequality, further research could include inequality estimations and decomposition analysis of health care utilisation data, such as paediatric emergency visits for asthma-related illness. This could give more insight into inequity in health care utilisation in the UK. It would also be interesting to apply this methodology to other countries for comparison. Questions validated by the ISAAC are commonly used in surveys from other countries, giving the opportunity to see how the UK performs alongside other countries.

7.8. Conclusion

The clear link between low income and adverse asthma-related outcomes has been confirmed by the findings of this thesis. In addition, my findings suggest that this relationship persists into late childhood (steepening of the child health-income gradient) in the case of the UK. Furthermore, this association persists when outcome measurements are adapted to reflect the heterogeneity of asthma by classification of wheezing symptoms. This thesis also provides novel evidence of income-inequality in asthma-related outcomes in children, indicating that asthma and wheezing are concentrated amongst children from low- income families in the UK. The decomposition analyses have highlighted that income itself is a major contributor to income-related inequality in asthma-related outcomes. By employing a variety of methodological approaches, this thesis makes a contribution to the methodology and the tools that can be applied to population health research.

In terms of policy, my results highlight how current reforms in the UK welfare system, such as cuts in benefits and the introduction of Universal Credit, may have a negative impact for many families and children. For a nation that already had a large proportion of its children living in low-income families, even before austerity measures were implemented from 2010, the UK is now at risk of reversing all the progress made towards childhood poverty elimination in the early 1990's. This thesis has showed that income is a crucial determinant of asthma-related outcomes in children. From a policy perspective, this evidence supports continued and improved welfare benefits for low-income families. Under the Health and Social Care Act of 2012, public health bodies in the UK have a statutory duty to reduce inequalities in health, and recommend a way to end income-related inequality in child health by tackling income discrepancies (Marmot, 2010). Therefore, limiting access to income from welfare, as has been done in the last ten years in the UK, is counter-intuitive to the countrywide goal to reduce income-related inequality in child health. It must also be noted that to tackle inequalities in asthma-related outcomes successfully, a multisectoral approach is required, with collaboration between key stakeholders (the government and society) and across sectors such as the health system (health access and availability), the environment and economic sectors.

Poor asthma-related outcomes in early childhood, later childhood and adolescence, may lead to long-term health issues, and can affect ones capacity to work in adulthood. From an economic perspective, not only will low income in childhood increase the risk of ill-health in adulthood, but it will also result in unhealthy adults in the future. In terms of fairness, reducing child poverty and health inequality is "a matter of fairness and social justice" , and if it not prioritised, society would have failed children by not giving them the best possible start in life (Marmot, 2010).

References

Allanson, P., Gerdtham, U.-G., Petrie, D., 2010. Longitudinal analysis of income-related health inequality. J Health Econ 29, 78–86. https://doi.org/10.1016/j.jhealeco.2009.10.005

Allin, S., Stabile, M., 2012. Socioeconomic status and child health: what is the role of health care, health conditions, injuries and maternal health? HEPL 7, 227–242. https://doi.org/10.1017/S174413311100034X

Andriessen, W., Brunekreef, B., Roemer,W., 1998. Home dampness and respiratory health status in European children: Home dampness and respiratory health. Clinical & Experimental Allergy 28, 1191–1200. https://doi.org/10.1046/j.1365-2222.1998.00322.x

Apouey, B., Geoffard, P.-Y., 2013. Family income and child health in the UK. Journal of Health Economics 32, 715–727. https://doi.org/10.1016/j.jhealeco.2013.03.006

Arathimos, R., Granell, R., Henderson, J., Relton, C.L., Tilling, K., 2017a. Sex discordance in asthma and wheeze prevalence in two longitudinal cohorts. PLoS ONE 12, e0176293. https://doi.org/10.1371/journal.pone.0176293

Asher, M.I., 2011. Urbanisation, asthma and allergies. Thorax 66, 1025–1026. https://doi.org/10.1136/thoraxjnl-2011-201019

Asthma UK. 2019. [WWW Document], 2016. URL https://www.asthma.org.uk/advice/understanding-asthma/what-is-asthma/

Azad, Meghan B., Konya, T., Maughan, H., Guttman, D.S., Field, C.J., Sears, M.R., Becker, A.B., Scott, J.A., Kozyrskyj, A.L., 2013. Infant gut microbiota and the hygiene hypothesis of allergic disease: impact of household pets and siblings on microbiota composition and diversity. Allergy Asthma Clin Immunol 9, 15. https://doi.org/10.1186/1710-1492-9-15

Azad, M.B., Vehling, L., Lu, Z., Dai, D., Subbarao, P., Becker, A.B., Mandhane, P.J., Turvey, S.E., Lefebvre, D.L., Sears, M.R., 2017. Breastfeeding, maternal asthma and wheezing in the first year of life: a longitudinal birth cohort study. Eur Respir J 49, 1602019. https://doi.org/10.1183/13993003.02019-2016

Baltagi, B.H., 1995. Econometric analysis of panel data. John Wiley & Sons, Chichester.

Baltagi, B.H., Lagravinese, R., Moscone, F., Tosetti, E., 2017. Health Care Expenditure and Income: A Global Perspective. Health Economics 26, 863–874. https://doi.org/10.1002/hec.3424

Becker, G.S., 1964. Human Capital: A Theoretical and Empirical Analysis, with Special Reference to Education (SSRN Scholarly Paper No. ID 1496221). Social Science Research Network, Rochester, NY.

Becker, G.S., Tomes, N., 1986. Human Capital and the Rise and Fall of Families. Journal of Labor Economics 4, S1–S39. https://doi.org/10.1086/298118

Belanger, K., Triche, E.W., 2008. Indoor combustion and asthma. Immunol Allergy Clin North Am 28, 507–519, vii. https://doi.org/10.1016/j.iac.2008.03.011

Belsky, J., Bell, B., Bradley, R.H., Stallard, N., Stewart-Brown, S.L., 2007. Socioeconomic risk, parenting during the preschool years and child health age 6 years. The European Journal of Public Health 17, 508–513. https://doi.org/10.1093/eurpub/ckl261

Berger, L.M., Paxson, C., Waldfogel, J., 2009. Income and child development. Children and Youth Services Review 31, 978–989. https://doi.org/10.1016/j.childyouth.2009.04.013

Blau, D.M., 1999. The Effect of Income on Child Development. Review of Economics and Statistics 81, 261–276. https://doi.org/10.1162/003465399558067

Boardman, J.D., Powers, D.A., Padilla, Y.C., Hummer, R.A., 2002. Low birth weight, social factors, and developmental outcomes among children in the United States. Demography 39, 353–368. https://doi.org/10.1353/dem.2002.0015

Brand, P.L.P., Baraldi, E., Bisgaard, H., Boner, A.L., Castro-Rodriguez, J.A., Custovic, A., de Blic, J., de Jongste, J.C., Eber, E., Everard, M.L., Frey, U., Gappa, M., Garcia-Marcos, L., Grigg, J., Lenney, W., Le Souef, P., McKenzie, S., Merkus, P.J.F.M., Midulla, F., Paton, J.Y., Piacentini, G., Pohunek, P., Rossi, G.A., Seddon, P., Silverman, M., Sly, P.D., Stick, S., Valiulis, A., van Aalderen, W.M.C., Wildhaber, J.H., Wennergren, G., Wilson, N., Zivkovic, Z., Bush, A., 2008. Definition, assessment and treatment of wheezing disorders in preschool children: an evidence-based approach. European Respiratory Journal 32, 1096–1110. https://doi.org/10.1183/09031936.00002108

Braun-Fahrländer, C., Riedler, J., Herz, U., Eder, W., Waser, M., Grize, L., Maisch, S., Carr, D., Gerlach, F., Bufe, A., Lauener, R.P., Schierl, R., Renz, H., Nowak, D., von Mutius, E., 2002. Environmental Exposure to Endotoxin and Its Relation to Asthma in School-Age Children. N Engl J Med 347, 869–877. https://doi.org/10.1056/NEJMoa020057

Brew, B.K., Chiesa, F., Lundholm, C., Örtqvist, A., Almqvist, C., 2019. A modern approach to identifying and characterizing child asthma and wheeze phenotypes based on clinical data. PLoS One 14, e0227091. https://doi.org/10.1371/journal.pone.0227091

Brewer, M., Browne, J., Joyce, R., Payne, J.K., Institute for Fiscal Studies (Great Britain), Economic and Social Research Council (Great Britain), 2011. Child and working-age poverty from 2010 to 2020. Institute for Fiscal Studies, London.

Cakmak, S., Hebbern, C., Cakmak, J.D., Vanos, J., 2016. The modifying effect of socioeconomic status on the relationship between traffic, air pollution and respiratory health in elementary schoolchildren. Journal of Environmental Management 177, 1–8. https://doi.org/10.1016/j.jenvman.2016.03.051

Case A, Lubotsky D, Paxson C. Economic Status and Health in Childhood: The Origins of the Gradient. Am Econ Rev. 2002;92(5):1308-34. doi: 10.1257/000282802762024520. PMID: 29058397

Case, A., Fertig, A., Paxson, C., 2005. The lasting impact of childhood health and circumstance. Journal of Health Economics 24, 365–389. https://doi.org/10.1016/j.jhealeco.2004.09.008

Case, A., Lee, D., Paxson, C., 2008. The income gradient in children's health: A comment on Currie, Shields and Wheatley Price. Journal of Health Economics 27, 801–807. https://doi.org/10.1016/j.jhealeco.2007.10.005

Chancel, L., 2019. Ten facts about income inequality in advanced economies (No. hal-02876982), Working Papers, Working Papers. HAL.

Chen, E., Martin, A.D., Matthews, K.A., 2006. Socioeconomic status and health: Do gradients differ within childhood and adolescence? Social Science & Medicine 62, 2161–2170. https://doi.org/10.1016/j.socscimed.2005.08.054

Chen, Y., Li, H., 2009. Mother's education and child health: Is there a nurturing effect? Journal of Health Economics 28, 413–426. https://doi.org/10.1016/j.jhealeco.2008.10.005

Child Poverty Action Group [WWW Document], n.d. . CPAG. URL https://cpag.org.uk/ (accessed 12.14.20).

Child Poverty Action Group. Child poverty facts and figures [WWW Document], 2016. CPAG. URL https://cpag.org.uk/child-poverty/child-poverty-facts-and-figures (accessed 12.14.20).

Child Poverty Action Group. The Austerity Generation: the impact of a decade of cuts on family incomes and child poverty [WWW Document], 2017. CPAG. URL https://cpag.org.uk/policy-and-campaigns/report/austerity-generation-impact-decade-cuts-family-incomes-and-child-poverty (accessed 1.17.21).

Choi, W.-J., Um, I.-Y., Hong, S., Yum, H.Y., Kim, H., Kwon, H., 2012a. Association between Household Income and Asthma Symptoms among Elementary School Children in Seoul. Environ Health Toxicol 27, e2012020. https://doi.org/10.5620/eht.2012.27.e2012020

Civelek, E., Cakir, B., Orhan, F., Yuksel, H., Boz, A.B., Uner, A., Sekerel, B.E., 2011. Risk factors for current wheezing and its phenotypes among elementary school children. Pediatric Pulmonology 46, 166–174. https://doi.org/10.1002/ppul.21346

Centre for Longitudinal Studies | 1970 British Cohort Study, n.d. URL https://cls.ucl.ac.uk/cls-studies/1970-british-cohort-study/ (accessed 1.22.21).

Condliffe, S., Link, C.R., 2008. The Relationship between Economic Status and Child Health: Evidence from the United States. American Economic Review 98, 1605–1618. https://doi.org/10.1257/aer.98.4.1605

Connelly, R., Platt, L., 2014. Cohort profile: UK Millennium Cohort Study (MCS). Int J Epidemiol 43, 1719–1725. https://doi.org/10.1093/ije/dyu001

Cookson, H., Granell, R., Joinson, C., Ben-Shlomo, Y., Henderson, A.J., 2009. Mothers' anxiety during pregnancy is associated with asthma in their children. Journal of Allergy and Clinical Immunology 123, 847-853.e11. https://doi.org/10.1016/j.jaci.2009.01.042

Cookson, R., Propper, C., Asaria, M., Raine, R., 2016a. Socio-Economic Inequalities in Health Care in England. Fiscal Studies 37, 371–403. https://doi.org/10.1111/j.1475-5890.2016.12109

Cooper, K., Stewart, K. Does Household Income Affect children's Outcomes? A Systematic Review of the Evidence. *Child Ind Res* (2020). https://doi.org/10.1007/s12187-020-09782-0

Costa Font, J., Hernández-Quevedo, C., McGuire, A., 2011. Persistence despite action? Measuring the patterns of health inequality in England (1997-2007). Health Policy 103, 149–159. https://doi.org/10.1016/j.healthpol.2011.07.002

Costa-Font, J., Hernández-Quevedo, C., 2012. Measuring inequalities in health: What do we know? What do we need to know? Health Policy 106, 195–206. https://doi.org/10.1016/j.healthpol.2012.04.007

Cowell, F., 2011. Measuring Inequality (OUP Catalogue). Oxford University Press.

Currie, A., Shields, M.A., Price, S.W., 2007. The child health/family income gradient: Evidence from England. Journal of Health Economics 26, 213–232. https://doi.org/10.1016/j.jhealeco.2006.08.003

Currie, J., 2009. Healthy, Wealthy, and Wise: Socioeconomic Status, Poor Health in Childhood, and Human Capital Development. Journal of Economic Literature 47, 87–122. https://doi.org/10.1257/jel.47.1.87

Currie, J., Stabile, M., 2003. Socioeconomic Status and Child Health: Why Is the Relationship Stronger for Older Children? American Economic Review 93, 1813–1823. https://doi.org/10.1257/000282803322655563

Darabi, B., Rahmati, S., HafeziAhmadi, M.R., Badfar, G., Azami, M., 2019. The association between caesarean section and childhood asthma: an updated systematic review and meta-analysis. Allergy, Asthma & Clinical Immunology 15, 62. https://doi.org/10.1186/s13223-019-0367-9

Deaton, A., 2003. Health, Inequality, and Economic Development. Journal of Economic Literature 41, 113–158. https://doi.org/10.1257/002205103321544710

Deliu, M., Belgrave, D., Sperrin, M., Buchan, I., Custovic, A., 2017a. Asthma phenotypes in childhood. Expert Rev Clin Immunol 13, 705–713. https://doi.org/10.1080/1744666X.2017.1257940

Department of Health and Social Care, 2019. The Government's revised mandate to NHS England for 2018-19. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/803111/revised-mandate-to-nhs-england-2018-to-2019.pdf

Deshpande, D., Morgan, W., 2016. Wheezing disorders in children: Are girls and boys different? Journal of Allergy and Clinical Immunology 138, 1569–1570. https://doi.org/10.1016/j.jaci.2016.07.034

Dogaru, C.M., Nyffenegger, D., Pescatore, A.M., Spycher, B.D., Kuehni, C.E., 2014. Breastfeeding and childhood asthma: systematic review and meta-analysis. Am J Epidemiol 179, 1153–1167. https://doi.org/10.1093/aje/kwu072

Doyle, O., Harmon, C., Walker, I. 2007. "The Impact of Parental Income and Education on Child Health : Further Evidence for England," The Warwick Economics Research Paper Series (TWERPS) 788, University of Warwick, Department of Economics.

El-Rachidi, S., LaRochelle, J.M., Morgan, J.A., 2017. Pharmacists and Pediatric Medication Adherence: Bridging the Gap. Hosp Pharm 52, 124–131. https://doi.org/10.1310/hpj5202-124

Erreygers, G., 2009. Correcting the concentration index. J Health Econ 28, 504–515. https://doi.org/10.1016/j.jhealeco.2008.02.003

Erreygers, G., Van Ourti, T., 2011. Measuring socioeconomic inequality in health, health care and health financing by means of rank-dependent indices: A recipe for good practice. Journal of Health Economics 30, 685–694. https://doi.org/10.1016/j.jhealeco.2011.04.004

Estrada, R.D., Ownby, D.R., 2017. Rural Asthma: Current Understanding of Prevalence, Patterns, and Interventions for Children and Adolescents. Curr Allergy Asthma Rep 17, 37. https://doi.org/10.1007/s11882-017-0704-3

Farfel, A., Tirosh, A., Derazne, E., Garty, B.Z., Afek, A., 2010. Association between socioeconomic status and the prevalence of asthma. Ann Allergy Asthma Immunol 104, 490–495. https://doi.org/10.1016/j.anai.2010.03.019

Fisk, W.J., Lei-Gomez, Q., Mendell, M.J., 2007. Meta-analyses of the associations of respiratory health effects with dampness and mold in homes. Indoor Air 17, 284–296. https://doi.org/10.1111/j.1600-0668.2007.00475.x

Fletcher, J., Wolfe, B., 2014. Increasing our understanding of the health-income gradient in children: gradient and health. Health Econ. 23, 473–486. https://doi.org/10.1002/hec.2969

Forno, E., Celedon, J.C., 2009. Asthma and ethnic minorities: socioeconomic status and beyond. Curr Opin Allergy Clin Immunol 9, 154–160. https://doi.org/10.1097/aci.0b013e3283292207

Francis-Devine, B., 2021. Poverty in the UK: statistics. https://commonslibrary.parliament.uk/research-briefings/sn07096/

Friedman, M., 2008. A theory of the consumption function, Reprint, 1. pbk. print. ed, National Bureau of economic research general series. Princeton Univ. Press, Princeton, NJ.

Galobardes, B., 2006. Indicators of socioeconomic position (part 1). Journal of Epidemiology & Community Health 60, 7–12. https://doi.org/10.1136/jech.2004.023531

Galobardes, B., Granell, R., Sterne, J., Hughes, R., Mejia-Lancheros, C., Davey Smith, G., Henderson, J., 2015a. Childhood Wheezing, Asthma, Allergy, Atopy, and Lung Function: Different Socioeconomic Patterns for Different Phenotypes. Am. J. Epidemiol. 182, 763–774. https://doi.org/10.1093/aje/kwv045

Ghaempanah, Z., Fazlollahi, M.R., Noorbala, A.A., Movahedi, M., Kazemnejad, A., Pourpak, Z., Moin, M., 2013. Impact of maternal mental health on pediatric asthma control. Tanaffos 12, 23–27.

Gondek, D., Ning, K., Ploubidis, G.B., Nasim, B., Goodman, A., 2018. The impact of health on economic and social outcomes in the United Kingdom: A scoping literature review. PLoS ONE 13, e0209659. https://doi.org/10.1371/journal.pone.0209659

Gong, T., Lundholm, C., Rejnö, G., Mood, C., Långström, N., Almqvist, C., 2014a. Parental Socioeconomic Status, Childhood Asthma and Medication Use – A Population-Based Study. PLoS ONE 9, e106579. https://doi.org/10.1371/journal.pone.0106579

Gosavi, S., 2016. Factors Contributing Towards Poor Asthma Control in Patients on Regular Medication. JCDR. https://doi.org/10.7860/JCDR/2016/18670.8052

Grossman, M., 2000a. Chapter 7 - The Human Capital Model* *I am indebted to Robert Kaestner, Sara Markowitz, Tomas Philipson, andWalter Ried for helpful comments., in: Culyer, A.J., Newhouse, J.P. (Eds.), Handbook of Health Economics, Handbook of Health Economics. Elsevier, pp. 347–408. https://doi.org/10.1016/S1574-0064(00)80166-3

Grossman, M., 2000b. Chapter 7 The human capital model, in: Handbook of Health Economics. Elsevier, pp. 347–408. https://doi.org/10.1016/S1574-0064(00)80166-3

Grossman, M., 1972. On the Concept of Health Capital and the Demand for Health. Journal of Political Economy 80, 223–255.

Hafkamp-de Groen, E., Sonnenschein-van der Voort, A.M.M., Mackenbach, J.P., Duijts, L., Jaddoe, V.W.V., Moll, H.A., Hofman, A., de Jongste, J.C., Raat, H., 2013. Socioeconomic and Sociodemographic Factors Associated with Asthma Related Outcomes in Early Childhood: The Generation R Study. PLoS ONE 8, e78266. https://doi.org/10.1371/journal.pone.0078266

Hafkamp-de Groen, E., van Rossem, L., de Jongste, J.C., Mohangoo, A.D., Moll, H.A., Jaddoe, V.W.V., Hofman, A., Mackenbach, J.P., Raat, H., 2012. The role of prenatal, perinatal and postnatal factors in the explanation of socioeconomic inequalities in preschool asthma symptoms: the Generation R Study. J Epidemiol Community Health 66, 1017–1024. https://doi.org/10.1136/jech-2011-200333

Hansen, K., 2014. Millennium Cohort Study. A Guide to the Datasets (Eighth Edition) First, Second, Third, Fourth and Fifth Surveys. Centre for Longitudinal Studies. https://cls.ucl.ac.uk/cls-studies/millennium-cohort-study/

Henderson J, Granell R, Heron J, Sherriff A, Simpson A, Woodcock A, Strachan DP, Shaheen SO, Sterne JA. Associations of wheezing phenotypes in the first 6 years of life with atopy, lung function and airway responsiveness in mid-childhood. Thorax. 2008 Nov;63(11):974-80. doi: 10.1136/thx.2007.093187. Epub 2008 Aug 4. PMID: 18678704; PMCID: PMC2582336.

Hernández-Quevedo C., Jones, A.M., López-Nicolás, A., Rice, N.,2006. Socioeconomic inequalities in health: A comparative longitudinal analysis using the European Community Household Panel. Social Science & Medicine 63, 1246-1261. https://doi.org/10.1016/j.socscimed.2006.03.017

Heshmati, A., 2006. The World Distribution of Income and Income Inequality: A Review of the Economics Literature*. JWSR 61–107. https://doi.org/10.5195/jwsr.2006.372

Hill, T.D., Davis, A.P., Roos, J.M., French, M.T., 2020. Limitations of Fixed-Effects Models for Panel Data. Sociological Perspectives 63, 357–369. https://doi.org/10.1177/0731121419863785

Hirsch, D. A Minimum Income Standard for the United Kingdom in 2019 [WWW Document], 2019. . JRF. URL https://www.jrf.org.uk/report/minimum-income-standard-uk-2019 (accessed 1.30.21).

Hong, J., Lee, J., 2019. Decomposing Income-Related Inequalities in Self-Reported Depression and Self-Rated Health Among Married Immigrants in South Korea. Int J Environ Res Public Health 16. https://doi.org/10.3390/ijerph16101869

Hood, A., Waters, T., 2017. Living standards, poverty and inequality in the UK: 2016–17 to 2021–22. The IFS. https://doi.org/10.1920/re.ifs.2017.0127

Hossny, E., Caraballo, L., Casale, T., El-Gamal, Y., Rosenwasser, L., 2017. Severe asthma and quality of life. World Allergy Organ J 10. https://doi.org/10.1186/s40413-017-0159-y

Hsiao, C. 2014. Analysis of Panel Data (3rd ed., Econometric Society Monographs). Cambridge: Cambridge University Press. doi:10.1017/CBO9781139839327

Ip, S., Chung, M., Raman, G., Trikalinos, T.A., Lau, J., 2009. A Summary of the Agency for Healthcare Research and Quality's Evidence Report on Breastfeeding in Developed Countries. Breastfeeding Medicine 4, S-17-S-30. https://doi.org/10.1089/bfm.2009.0050

Iqbal, S.M., 2012. Managing wheeze in preschool children: How difficult can it be? Sudan J Paediatr 12, 17–26.

Johnson, R.C., Schoeni, R.F., 2011. The Influence of Early-Life Events on Human Capital, Health Status, and Labor Market Outcomes Over the Life Course. The B.E. Journal of Economic Analysis & Policy 11. https://doi.org/10.2202/1935-1682.2521

Jones, A.M., 2009. Panel Data Methods and Applications to Health Economics, in: Mills, T.C., Patterson, K. (Eds.), Palgrave Handbook of Econometrics. Palgrave Macmillan UK, London, pp. 557–631. https://doi.org/10.1057/9780230244405_12

Judge, L., 2012. Ending child poverty by 2020: progress made and lessons learned. Child Poverty Action Group, London.

Kakwani, N., Wagstaff, A., van Doorslaer, E., 1997. Socioeconomic inequalities in health: Measurement, computation, and statistical inference. Journal of Econometrics 77, 87–103. https://doi.org/10.1016/S0304-4076(96)01807-6

Kato, T., Yorifuji, T., Yamakawa, M., Inoue, S., Doi, H., Eboshida, A., Kawachi, I., 2017. Association of maternal age with child health: A Japanese longitudinal study. PLoS One 12. https://doi.org/10.1371/journal.pone.0172544

Kavanagh, J., Jackson, D.J., Kent, B.D., 2019. Over- and under-diagnosis in asthma. Breathe 15, e20–e27. https://doi.org/10.1183/20734735.0362-2018

Keag, O.E., Norman, J.E., Stock, S.J., 2018. Long-term risks and benefits associated with cesarean delivery for mother, baby, and subsequent pregnancies: Systematic review and meta-analysis. PLoS Med 15, e1002494. https://doi.org/10.1371/journal.pmed.1002494

Kelly, Y.J., Sacker, A., Gray, R., Kelly, J., Wolke, D., Head, J., Quigley, M.A., 2012. Light drinking during pregnancy: still no increased risk for socioemotional difficulties or cognitive deficits at 5 years of age? J Epidemiol Community Health 66, 41–48. https://doi.org/10.1136/jech.2009.103002

Kennedy, S., 2014.Child Poverty Act 2010: a short guide. https://commonslibrary.parliament.uk/research-briefings/sn05585/

Kennedy, S., 2012. Universal Credit: an introduction. https://commonslibrary.parliament.uk/research-briefings/sn06469/

Kessler, R.C., Andrews, G., Colpe, L.J., Hiripi, E., Mroczek, D.K., Normand, S.L.T., Walters, E.E., Zaslavsky, A.M., 2002. Short screening scales to monitor population prevalences and trends in non-specific psychological distress. Psychol Med 32, 959–976. https://doi.org/10.1017/s0033291702006074

Khanam, R., Nghiem, H.S., Connelly, L.B., 2009a. Child health and the income gradient: Evidence from Australia. Journal of Health Economics 28, 805–817. https://doi.org/10.1016/j.jhealeco.2009.05.001

Kjellsson, G., Gerdtham, U.-G., 2013. On correcting the concentration index for binary variables. J Health Econ 32, 659–670. https://doi.org/10.1016/j.jhealeco.2012.10.012

Krieger, J., 2010. Home is Where the Triggers Are: Increasing Asthma Control by Improving the Home Environment. Pediatr Allergy Immunol Pulmonol 23, 139–145. https://doi.org/10.1089/ped.2010.0022

Kruk, K.E., 2013. Parental income and the dynamics of health inequality in early childhood-evidence from the UK: parental income and the dynamics of health inequality in childhood. Health Econ. 22, 1199–1214. https://doi.org/10.1002/hec.2876

Kuehnle D. The causal effect of family income on child health in the U.K. J Health Econ. 2014 Jul;36:137-50. doi: 10.1016/j.jhealeco.2014.03.011. Epub 2014 Apr 13. PMID: 24794502.

Kurukulaaratchy, R.J., Fenn, M.H., Waterhouse, L.M., Matthews, S.M., Holgate, S.T., Arshad, S.H., 2003. Characterization of wheezing phenotypes in the first 10 years of life. Clin Exp Allergy 33, 573–578. https://doi.org/10.1046/j.1365-2222.2003.01657.x

Laerum, B.N., Svanes, C., Wentzel-Larsen, T., Gulsvik, A., Torén, K., Norrman, E., Gíslason, T., Janson, C., Omenaas, E., 2007. Young maternal age at delivery is associated with asthma in adult offspring. Respir Med 101, 1431–1438. https://doi.org/10.1016/j.rmed.2007.01.020

Lawson, J.A., Rennie, D.C., Cockcroft, D.W., Dyck, R., Afanasieva, A., Oluwole, O., Afsana, J., 2017. Childhood asthma, asthma severity indicators, and related conditions along an urban-rural gradient: a cross-sectional study. BMC Pulm Med 17. https://doi.org/10.1186/s12890-016-0355-5

Lewis, J., Lister, R., Millar, J., 2001. Ending child poverty: what is happening in the uk? [WWW document], URL https://www.issuelab.org/resources/357/357.pdf (accessed 28.12.2020)

Lewis, K.M., Ruiz, M., Goldblatt, P., Morrison, J., Porta, D., Forastiere, F., Hryhorczuk, D., Zvinchuk, O., Saurel-Cubizolles, M.-J., Lioret, S., Annesi-Maesano, I., Vrijheid, M., Torrent, M., Iniguez, C., Larranaga, I., Harskamp-van Ginkel, M.W., Vrijkotte, T.G.M., Klanova, J., Svancara, J., Barross, H., Correia, S., Jarvelin, M.-R., Taanila, A., Ludvigsson, J., Faresjo, T., Marmot, M., Pikhart, H., 2017. Mother's education and offspring asthma risk in 10 European cohort studies. Eur J Epidemiol 32, 797–805. https://doi.org/10.1007/s10654-017-0309-0

Lim, R.H., Kobzik, L., Dahl, M., 2010. Risk for asthma in offspring of asthmatic mothers versus fathers: a meta-analysis. PLoS One 5, e10134. https://doi.org/10.1371/journal.pone.0010134

Lodge, C.J., Zaloumis, S., Lowe, A.J., Gurrin, L.C., Matheson, M.C., Axelrad, C., Bennett, C.M., Hill, D.J., Hosking, C.S., Svanes, C., Abramson, M.J., Allen, K.J., Dharmage, S.C., 2014. Early-life risk factors for childhood wheeze phenotypes in a high-risk birth cohort. J Pediatr 164, 289-294.e1–2. https://doi.org/10.1016/j.jpeds.2013.09.056

Lowe, L.A., Simpson, A., Woodcock, A., Morris, J., Murray, C.S., Custovic, A., NAC Manchester Asthma and Allergy Study Group, 2005. Wheeze phenotypes and lung function in preschool children. Am J Respir Crit Care Med 171, 231–237. https://doi.org/10.1164/rccm.200406-695OC

Magnus, M.C., Håberg, S.E., Stigum, H., Nafstad, P., London, S.J., Vangen, S., Nystad, W., 2011a. Delivery by Cesarean Section and Early Childhood Respiratory Symptoms and Disorders. Am J Epidemiol 174, 1275–1285. https://doi.org/10.1093/aje/kwr242

Magnus MC, DeRoo LA, Håberg SE, Magnus P, Nafstad P, Nystad W, London SJ. Prospective study of maternal alcohol intake during pregnancy or lactation and risk of childhood asthma: the Norwegian Mother and Child Cohort Study. Alcohol Clin Exp Res. 2014 Apr;38(4):1002-11. doi: 10.1111/acer.12348.

Malat, J., Oh, H.J., Hamilton, M.A., 2005. Poverty Experience, Race, and Child Health. Public Health Rep 120, 442–447. https://doi.org/10.1177/003335490512000411

Malik, H.U.-R., Kumar, K., Frieri, M., 2012. Minimal Difference in the Prevalence of Asthma in the Urban and Rural Environment. Clin Med Insights Pediatr 6, 33–39. https://doi.org/10.4137/CMPed.S9539

Marmot, M., 2010. Fair Society Healthy Lives (The Marmot Review) - IHE [WWW Document], n.d. URL http://www.instituteofhealthequity.org/resources-reports/fair-society-healthy-lives-the-marmot-review (accessed 1.31.21).

Marmot, M., 2020. Health equity in England: the Marmot review 10 years on. BMJ 368, m693. https://doi.org/10.1136/bmj.m693

Martinez, F.D., Wright, A.L., Taussig, L.M., Holberg, C.J., Halonen, M., Morgan, W.J., 1995. Asthma and wheezing in the first six years of life. The Group Health Medical Associates. N Engl J Med 332, 133–138. https://doi.org/10.1056/NEJM199501193320301

Mayer, S.E., New Zealand, Ministry of Social Development, Knowledge Management Group, 2002. The influence of parental income on children's outcomes. Knowledge Management Group, Ministry of Social Development, Wellington [N.Z.

Maykut, R., Zeiger, R.S., Brown, M.A., 2010. Asthma in pediatric patients: unmet need and therapeutic options. Clin Pediatr (Phila) 49, 915–930. https://doi.org/10.1177/0009922810362590

McCartney, G., Popham, F., McMaster, R., Cumbers, A., 2019. Defining health and health inequalities. Public Health 172, 22–30. https://doi.org/10.1016/j.puhe.2019.03.023

Millenium Cohort Study. 2021. Centre for Longitudinal Studies. https://cls.ucl.ac.uk/cls-studies/millennium-cohort-study/

Microeconometrics: Methods and Applications by A. Colin Cameron and P.K. Trivedi [WWW Document], n.d. URL http://cameron.econ.ucdavis.edu/mmabook/mmadata.html (accessed 12.17.20).

Midodzi, W.K., Rowe, B.H., Majaesic, C.M., Saunders, L.D., Senthilselvan, A., 2008. Predictors for wheezing phenotypes in the first decade of life. Respirology 13, 537–545. https://doi.org/10.1111/j.1440-1843.2008.01284.x

Midouhas, E., Kokosi, T., Flouri, E., 2019a. The quality of air outside and inside the home: associations with emotional and behavioural problem scores in early childhood. BMC Public Health 19, 406. https://doi.org/10.1186/s12889-019-6733-1

Mitchell, C.M., Beals, J., 2011. The utility of the Kessler Screening Scale for Psychological Distress (K6) in two American Indian communities. Psychol Assess 23, 752–761. https://doi.org/10.1037/a0023288

Miyake, Y., Tanaka, K., Sasaki, S., Kiyohara, C., Ohya, Y., Fukushima, W., Yokoyama, T., Hirota, Y., Osaka Maternal and Child Health Study Group, 2008. Breastfeeding and the risk of wheeze

and asthma in Japanese infants: the Osaka Maternal and Child Health Study. Pediatr Allergy Immunol 19, 490–496. https://doi.org/10.1111/j.1399-3038.2007.00701.x

Morris, J.N., Donkin, A.J., Wonderling, D., Wilkinson, P., Dowler, E.A., 2000. A minimum income for healthy living. J Epidemiol Community Health 54, 885–889. https://doi.org/10.1136/jech.54.12.885

Mukherjee, M., Stoddart, A., Gupta, R.P., Nwaru, B.I., Farr, A., Heaven, M., Fitzsimmons, D., Bandyopadhyay, A., Aftab, C., Simpson, C.R., Lyons, R.A., Fischbacher, C., Dibben, C., Shields, M.D., Phillips, C.J., Strachan, D.P., Davies, G.A., McKinstry, B., Sheikh, A., 2016a. The epidemiology, healthcare and societal burden and costs of asthma in the UK and its member nations: analyses of standalone and linked national databases. BMC Medicine 14, 113. https://doi.org/10.1186/s12916-016-0657-8

Nasim, B., 2015. Changes in the relationship between social housing tenure and child outcomes over time: Comparing the Millennium and British Cohort Studies (No. 15– 06), DoQSS Working Papers, DoQSS Working Papers. Quantitative Social Science - UCL Social Research Institute, University College London.

National Research Council (US) and Institute of Medicine (US). 2004. Children's Health, the Nation's Wealth: Assessing and Improving Child Health. Committee on Evaluation of Children's Health. Board on Children, Youth, and Families, Division of Behavioral and Social Sciences and Education. Washington, DC: The National Academies Press.

National Research Council (US) and Institute of Medicine (US) Committee on Depression, Parenting Practices, and the Healthy Development of Children; England MJ, Sim LJ, editors. Depression in Parents, Parenting, and Children: Opportunities to Improve Identification, Treatment, and Prevention. Washington (DC): National Academies Press (US); 2009. Available from: https://www.ncbi.nlm.nih.gov/books/NBK215117/ doi: 10.17226/12565

Neuman, Å., Hohmann, C., Orsini, N., Pershagen, G., Eller, E., Kjaer, H.F., Gehring, U., Granell, R., Henderson, J., Heinrich, J., Lau, S., Nieuwenhuijsen, M., Sunyer, J., Tischer, C., Torrent, M., Wahn, U., Wijga, A.H., Wickman, M., Keil, T., Bergström, A., ENRIECO Consortium, 2012. Maternal smoking in pregnancy and asthma in preschool children: a pooled analysis of eight birth cohorts. Am J Respir Crit Care Med 186, 1037–1043. https://doi.org/10.1164/rccm.201203-0501OC

Nuttall, F.Q., 2015. Body Mass Index: Obesity, BMI, and Health: A Critical Review. Nutr Today 50, 117–128. https://doi.org/10.1097/NT.0000000000000092

O'Donnell, O., Doorslaer, E. van, Wagstaff, A., Lindelow, M., 2008. Analyzing Health Equity Using Household Survey Data : A Guide to Techniques and Their Implementation, World Bank Publications. The World Bank.

O'Donnell, O., O'Neill, S., Van Ourti, T., Walsh, B., 2016. Conindex: Estimation of Concentration Indices. The Stata Journal 16, 112–138. https://doi.org/10.1177/1536867X1601600112

OECD.2021.Poverty. URL https://www.oecd-ilibrary.org/sites/8483c82f-en/index.html?itemId=/content/component/8483c82f-en#:~:text=People%20are%20classified%20as%20poor,have%20the%20higher%20poverty%20thresholds. (Accessed on 01 February 2021)

OECD. 2021. Income inequality (indicator). doi: 10.1787/459aa7f1-en (Accessed on 01 February 2021)

Panico, L., Stuart, B., Bartley, M., Kelly, Y., 2014. Asthma Trajectories in Early Childhood: Identifying Modifiable Factors. PLoS ONE 9, e111922. https://doi.org/10.1371/journal.pone.0111922

Parkin, E., 2021. Mental health policy in England. House of Commons Library briefing on mental health policy in England. https://commonslibrary.parliament.uk/research-briefings/cbp-7547/

Platt, L., Centre for Longitudinal Studies (London, E., 2014. Millennium Cohort Study: initial findings from the age 11 survey. Centre for Longitudinal Studies, Institution of Education, London.

Plewis, I., Kallis, C., 2008. Changing economic circumstances in childhood and their effects on susbsequent educational and other outcomes (No. Working Paper No 49). Department for Work and Pensions.

Pokhrel, S, Quigley, M.A., Fox-Rushby, J., McCormick, F., Williams, A., Trueman, P., Dodds, R., Renfrew, M.J., 2015. Potential economic impacts from improving breastfeeding rates in the UK. Arch Dis Child 100, 334–340. https://doi.org/10.1136/archdischild-2014-306701

Polinski, K.J., Liu, J., Boghossian, N.S., McLain, A.C., 2017. Maternal Obesity, Gestational Weight Gain, and Asthma in Offspring. Prev Chronic Dis 14, E109. https://doi.org/10.5888/pcd14.170196

Propper, Carol and Rigg, John, Understanding Socio-Economic Inequalities in Childhood Respiratory Health (March 2006). LSE STICERD Research Paper No. CASE109, Available at SSRN: https://ssrn.com/abstract=1159340

Propper, C., Rigg, J., Burgess, S., 2007. Child health: evidence on the roles of family income and maternal mental health from a UK birth cohort. Health Econ. 25.

Public Health England.[WWW Document], 2015. URL https://www.gov.uk/government/publications/local-action-on-health-inequalities-improving-health-literacy (accessed on 2.01.2021)

Quigley, M., McGuire, W., 2014. Formula versus donor breast milk for feeding preterm or low birth weight infants. Cochrane Database Syst Rev CD002971. https://doi.org/10.1002/14651858.CD002971.pub3

Quigley, M.A., Carson, C., Kelly, Y., 2018. Breastfeeding and Childhood Wheeze: Age-Specific Analyses and Longitudinal Wheezing Phenotypes as Complementary Approaches to the Analysis of Cohort Data. American Journal of Epidemiology 187, 1651–1661. https://doi.org/10.1093/aje/kwy057

Reeves, A., 2017. Reductions in housing benefit increases depression risk in low-income UK householdsAaron Reeves. Eur J Public Health 27. https://doi.org/10.1093/eurpub/ckx187.819

Reinhold, S., Jürges, H., 2012. Parental income and child health in Germany. Health Economics 21, 562–579. https://doi.org/10.1002/hec.1732

Ritz, T., Meuret, A.E., Trueba, A.F., Fritzsche, A., von Leupoldt, A., 2013. Psychosocial factors and behavioral medicine interventions in asthma. J Consult Clin Psychol 81, 231–250. https://doi.org/10.1037/a0030187

Rollins, Nigel C, Bhandari, N., Hajeebhoy, N., Horton, S., Lutter, C.K., Martines, J.C., Piwoz, E.G., Richter, L.M., Victora, C.G., 2016. Why invest, and what it will take to improve breastfeeding practices? The Lancet 387, 491–504. https://doi.org/10.1016/S0140-6736(15)01044-2

Royal College of Obstetricians and Gynaecologists. Key recommendations [WWW Document], n.d. URL https://www.rcog.org.uk/en/news/campaigns-and-opinions/better-for-women/key-recommendations/ (accessed 1.31.21).

Royal College of Paediatrics and Child Health. State of Child Health – Insight into the state of child health in the UK [WWW Document], n.d. URL https://stateofchildhealth.rcpch.ac.uk/ (accessed 1.31.21).

Rusconi, F., Galassi, C., Corbo, G.M., Forastiere, F., Biggeri, A., Ciccone, G., Renzoni, E., 1999a. Risk factors for early, persistent, and late-onset wheezing in young children. SIDRIA Collaborative Group. Am J Respir Crit Care Med 160, 1617–1622. https://doi.org/10.1164/ajrccm.160.5.9811002

Savenije, O.E., Granell, R., Caudri, D., Koppelman, G.H., Smit, H.A., Wijga, A., de Jongste, J.C., Brunekreef, B., Sterne, J.A., Postma, D.S., Henderson, J., Kerkhof, M., 2011a. Comparison of childhood wheezing phenotypes in 2 birth cohorts: ALSPAC and PIAMA. J Allergy Clin Immunol 127, 1505-1512.e14. https://doi.org/10.1016/j.jaci.2011.02.002

Sears, M.R., Greene, J.M., Willan, A.R., Wiecek, E.M., Taylor, D.R., Flannery, E.M., Cowan, J.O., Herbison, G.P., Silva, P.A., Poulton, R., 2003. A longitudinal, population-based, cohort study of childhood asthma followed to adulthood. N Engl J Med 349, 1414–1422. https://doi.org/10.1056/NEJMoa022363

Sheikh, A., Steiner, M.F.C., Cezard, G., Bansal, N., Fischbacher, C., Simpson, C.R., Douglas, A., Bhopal, R., on behalf of the SHELS researchers, 2016a. Ethnic variations in asthma hospital

admission, readmission and death: a retrospective, national cohort study of 4.62 million people in Scotland. BMC Medicine 14, 3. https://doi.org/10.1186/s12916-015-0546-6

Spycher, B.D., Silverman, M., Brooke, A.M., Minder, C.E., Kuehni, C.E., 2008. Distinguishing phenotypes of childhood wheeze and cough using latent class analysis. Eur Respir J 31, 974–981. https://doi.org/10.1183/09031936.00153507

Starr, M.A., 2014. Qualitative and mixed-methods research in economics: surprising growth, promising future. Journal of Economic Surveys 28, 238–264. https://doi.org/10.1111/joes.12004

Stata: Software for Statistics and Data Science [WWW Document], n.d. URL https://www.stata.com/ (accessed 1.24.21).

Subbarao, P., Mandhane, P.J., Sears, M.R., 2009. Asthma: epidemiology, etiology and risk factors. Canadian Medical Association Journal 181, E181–E190. https://doi.org/10.1503/cmaj.080612

Szumilas, M., 2010. Explaining Odds Ratios. J Can Acad Child Adolesc Psychiatry 19, 227–229.

Taylor-Robinson, D.C., Pearce, A., Whitehead, M., Smyth, R., Law, C., 2015. Social inequalities in wheezing in children: findings from the UK Millennium Cohort Study. Eur Respir J 47, 818–828. https://doi.org/10.1183/13993003.01117-2015

Thacher, J.D., Gruzieva, O., Pershagen, G., Melén, E., Lorentzen, J.C., Kull, I., Bergström, A., 2017. Mold and dampness exposure and allergic outcomes from birth to adolescence: data from the BAMSE cohort. Allergy 72, 967–974. https://doi.org/10.1111/all.13102

Tinuoye, O., Pell, J.P., Mackay, D.F., 2013. Meta-analysis of the association between secondhand smoke exposure and physician-diagnosed childhood asthma. Nicotine Tob Res 15, 1475–1483. https://doi.org/10.1093/ntr/ntt033

Ullemar, V., Magnusson, P.K.E., Lundholm, C., Zettergren, A., Melén, E., Lichtenstein, P., Almqvist, C., 2016. Heritability and confirmation of genetic association studies for childhood asthma in twins. Allergy 71, 230–238. https://doi.org/10.1111/all.12783

van Aalderen, W.M., 2012. Childhood Asthma: Diagnosis and Treatment. Scientifica (Cairo) 2012. https://doi.org/10.6064/2012/674204

Van de Poel, E., Van Doorslaer, E., O'Donnell, O., 2012. Measurement of inequity in health care with heterogeneous response of use to need. Journal of Health Economics 31, 676–689. https://doi.org/10.1016/j.jhealeco.2012.05.005

van den Bemt, L., Kooijman, S., Linssen, V., Lucassen, P., Muris, J., Slabbers, G., Schermer, T., 2010. How does asthma influence the daily life of children? Results of focus group interviews. Health Qual Life Outcomes 8, 5. https://doi.org/10.1186/1477-7525-8-5

van Doorslaer, E., Koolman, X., 2004. Explaining the differences in income-related health inequalities across European countries [WWW Document]. Health economics. https://doi.org/10.1002/hec.918

van Roode, T., Sharples, K., Dickson, N., Paul, C., 2017. Life-Course Relationship between Socioeconomic Circumstances and Timing of First Birth in a Birth Cohort. PLOS ONE 12, e0170170. https://doi.org/10.1371/journal.pone.0170170

Villamor, E., Iliadou, A., Cnattingius, S., 2009. Is the association between low birth weight and asthma independent of genetic and shared environmental factors? Am J Epidemiol 169, 1337–1343. https://doi.org/10.1093/aje/kwp054

Violato, M., Petrou, S., Gray, R., 2009. The relationship between household income and childhood respiratory health in the United Kingdom. Social Science & Medicine 69, 955–963. https://doi.org/10.1016/j.socscimed.2009.06.036

Violato, M., Petrou, S., Gray, R., Redshaw, M., 2011. Family income and child cognitive and behavioural development in the United Kingdom: does money matter? Health Econ. 20, 1201–1225. https://doi.org/10.1002/hec.1665

Wagstaff, A., 2009. Correcting the concentration index: a comment. J Health Econ 28, 516–520, author reply 521-524. https://doi.org/10.1016/j.jhealeco.2008.12.003

Wagstaff, A., 2005. The bounds of the concentration index when the variable of interest is binary, with an application to immunization inequality. Health Econ 14, 429–432. https://doi.org/10.1002/hec.953

Wagstaff, A., Paci, P., van Doorslaer, E., 1991. On the measurement of inequalities in health. Soc Sci Med 33, 545–557. https://doi.org/10.1016/0277-9536(91)90212-u

Wagstaff, A., van Doorslaer, E., Watanabe, N., 2003. On decomposing the causes of health sector inequalities with an application to malnutrition inequalities in Vietnam. Journal of Econometrics, Analysis of data on health: 2 112, 207–223. https://doi.org/10.1016/S0304-4076(02)00161-6

Walsh, B., Cullinan, J., 2015. Decomposing socioeconomic inequalities in childhood obesity: evidence from Ireland. Econ Hum Biol 16, 60–72. https://doi.org/10.1016/j.ehb.2014.01.003

Wenzel, S.E., 2012. Asthma phenotypes: the evolution from clinical to molecular approaches. Nat Med 18, 716–725. https://doi.org/10.1038/nm.2678

Welfare Reform and Work Act 2016. [WWW document], URL https://www.legislation.gov.uk/ukpga/2016/7/introduction/enacted (accessed 23.11.2020)

Whitrow, M.J., Harding, S., 2010. Asthma in Black African, Black Caribbean and South Asian adolescents in the MRC DASH study: a cross sectional analysis. BMC Pediatr 10, 18. https://doi.org/10.1186/1471-2431-10-18

Wickham S, Anwar E, Barr B, Law C, Taylor-Robinson D. Poverty and child health in the UK: using evidence for action. Arch Dis Child. 2016 Aug;101(8):759-66. doi: 10.1136/archdischild-2014-306746

Wooldridge, J., 2002. Econometric Analysis of Cross Section and Panel Data. The MIT Press, Cambridge, Masschusetts.

WHO | The World Health Organization's infant feeding recommendation [WWW Document], n.d. . WHO. URL https://www.who.int/nutrition/topics/infantfeeding_recommendation/en/ (accessed 1.31.21).

WHO | Too many babies are born too small [WWW Document], n.d. URL https://www.who.int/news/item/16-05-2019-too-many-babies-are-born-too-small (accessed 1.23.21).

Zacharasiewicz, A., 2016. Maternal smoking in pregnancy and its influence on childhood asthma. ERJ Open Res 2. https://doi.org/10.1183/23120541.00042-2016